

ABSTRACT

OF

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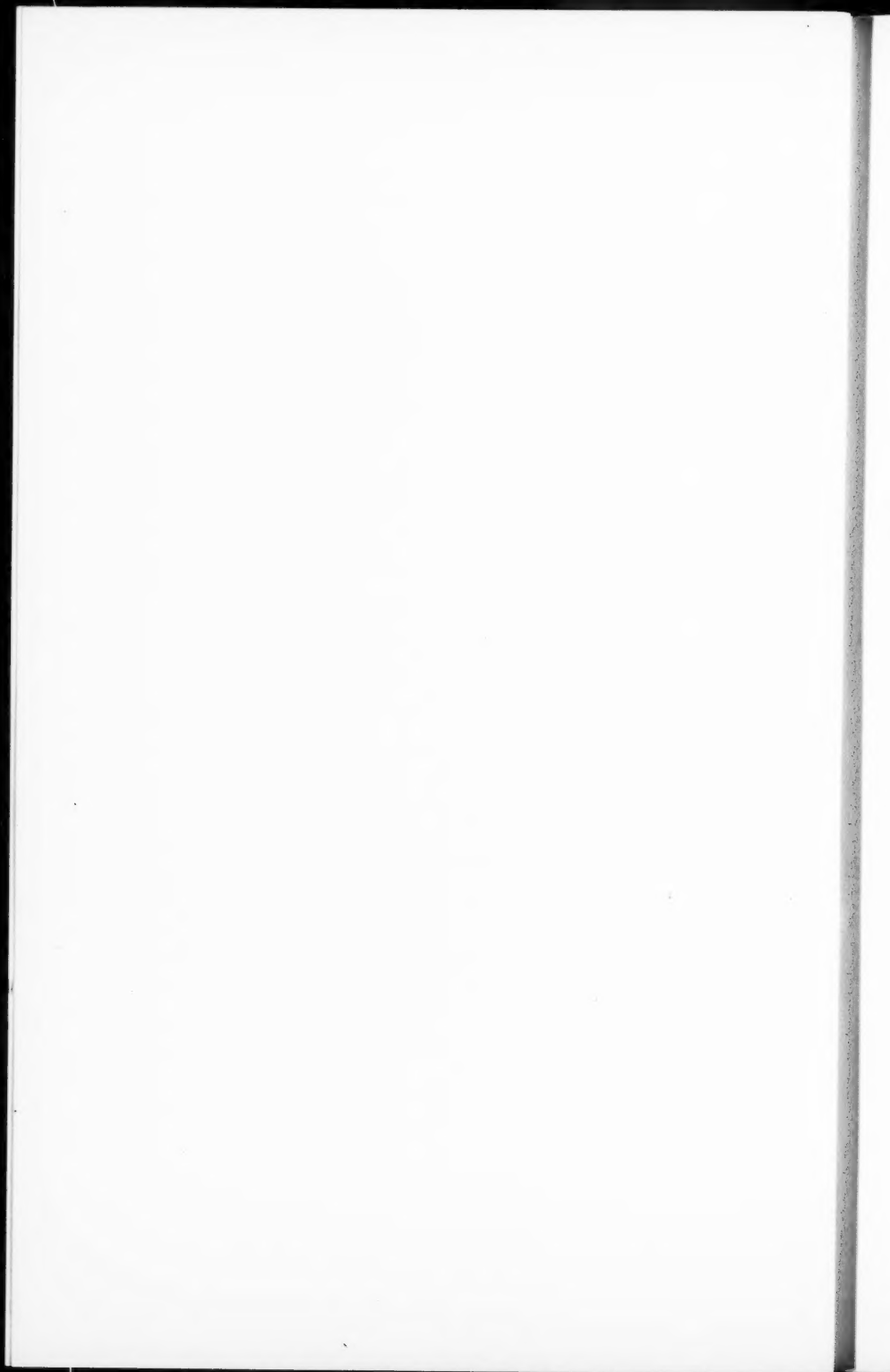
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**THE ASSOCIATION OF LIFE INSURANCE
MEDICAL DIRECTORS OF AMERICA**

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An Abstract of the Proceedings
OF THE
Association of
Life Insurance Medical Directors
of America

FORTY-FOURTH ANNUAL MEETING

The Forty-fourth Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Royal York Hotel, Toronto, Canada, on October 12 and 13, 1933.

The following members, delegates and guests were present at some time during the sessions: Doctors J. W. Abbott, G. E. Allen, H. B. Anderson, T. D. Archibald, W. B. Aten, A. E. Awde, H. A. Baker, Lewellys Barker, W. B. Bartlett, J. T. J. Battle, W. W. Beckett, M. B. Bander, E. B. Bigelow, C. C. Birchard, W. F. Blackford, J. C. Boland, William Bolt, J. T. Bowman, F. G. Brathwaite, H. B. Brown, B. F. Byrd, W. H. Carpenter, H. R. Carstens, A. H. W. Caulfeild, C. P. Clark, H. W. Cook, D. B. Cragin, H. C. Cruikshank, R. M. Daley, A. C. DeGraff, H. D. Delamere, E. J. Dewess, E. G. Dewis, W. W. Dinsmore, P. G. Drake, J. T. Eads, O. M. Eakins, J. H. Elliott, W. G. Exton, J. G. Falconer, H. H. Fellows, John Ferguson, W. E. Ferguson, J. W. Fisher, Otto Folin, R. A. Fraser, H. M. Frost, A. H. Gordon, Angus Graham, Harris Gray, F. R. Griffin, F. L. Grosvenor, Llewellyn Hall, Frank Harnden, H. M. Harrison, C. A. Heiken, E. M. Henderson, J. Hepburn, W. L. Hilliard, J. H. Holbrook, W. G. Hutchinson, H. S. Hutchison, W. A. Jaquith, A. O. Jimenis, A. E. Johann, A. S. Knight, E. H.

Lindstrom, J. M. Livingston, G. W. Lougheed, Carl Lovelace, C. L. McCloud, G. G. McConnell, F. H. McCrudden, C. B. McCulloch, A. A. MacDonald, L. F. MacKenzie, A. L. MacKinnon, George McLean, W. T. McNaughton, Charles Mærtz, R. W. Mann, W. L. Mann, L. K. Meredith, R. C. Montgomery, E. B. Mountain, William Muhlberg, R. W. Naylor, J. B. Neal, C. T. Necker, J. B. Nichols, E. C. Noble, James Norman, Herbert Old, M. I. Olsen, H. E. B. Pardee, H. C. Parsons, C. A. Peters, J. S. Phelps, C. B. Piper, I. M. Rabinowitch, J. A. Roberts, A. J. Robinson, F. W. Rolph, Gordon Ross, R. M. Rowley, W. W. Rucks, C. L. Rudasill, S. C. Rumford, E. F. Russell, H. C. Scadding, S. B. Scholz, Jr., B. T. D. Schwarz, H. N. Segall, J. T. Sheridan, R. L. Shields, J. L. Siner, V. F. Stock, D. D. Stofer, A. C. Stokes, A. R. Stone, S. J. Streight, L. G. Sykes, H. F. Taylor, W. A. Thomson, W. E. Thornton, Joseph Travenick, Wallace Troup, F. L. Truitt, H. B. Turner, J. P. Turner, H. E. Ungerleider, R. W. Urquhart, W. R. Ward, F. S. Weisse, F. L. Wells, C. D. Wheeler, C. F. S. Whitney, O. B. Wight, A. B. Wilkes, M. C. Wilson, G. E. Woodford, L. S. Ylvisaker, A. W. Young, and Messrs. J. H. Birkenshaw, R. G. Clarke, L. I. Dublin, J. W. Fisher, R. E. Fletcher, Arthur Hunter, H. J. Johnson, G. C. Kingsley, Leighton McCarthy, and J. G. Parker.

Total attendance at all sessions, 153.

During the past year, one member of our Association has died. The following testimonial to his memory has been prepared by representatives of the Association.

DR. SAMUEL WESLEY GADD

It is with sincere regret that I bring to your attention the death, on April 10, 1933, of Dr. Samuel Wesley Gadd, Medical Director of the Philadelphia Life Insurance Company and a member of this Association.

Dr. Gadd was born on November 19, 1859, at Llangrove, Herefordshire, England, the youngest of a large family. His

father was a lay-preacher in the Methodist Circuit in the Shire. When he was twelve years of age he migrated to America with his Mother and Father and joined one of his brothers, who had migrated a few years previously. They went to Louisville, Kentucky, but things did not go so well, so as they heard you could pick up gold in the streets of Philadelphia, they removed to Philadelphia. He tried to enter the Central High School, but because it was late after the school term had begun, the principal would not let him enter that year. He had a letter of introduction to a jeweler on Chestnut Street from a jeweler in Louisville, for whom he had worked as an errand boy, and the jeweler in Philadelphia took him on working for him.

Later, Dr. Kredick, who owned a drug store in South Philadelphia here where they were living took a liking to the boy and he went to work for him. He attended the Philadelphia College of Pharmacy and worked in Dr. Kredick's drug store at night, graduating with honors in the class of 1880. He acquired his own drug store at 8th and Federal Streets and it has been said that he was the first druggist in Philadelphia to serve ice cream sodas and was called crazy for doing it. He graduated from the University of Pennsylvania Medical School in the class of 1885.

He was at one time one of the examiners for the John Hancock Mutual Life Insurance Company and Medical Examiner-in-Chief of the Artisans Order of Mutual Protection, as well as an examiner for the Philadelphia Life Insurance Company. Upon the death of Dr. T. Hewson Bradford, Medical Director of the Philadelphia Life Insurance Company, in June, 1915, he was appointed by the Company to succeed him, whereupon he gave up his medical practice and other interests and devoted his time to serving the Philadelphia Life Insurance Company until his death.

He was one of the founders of the Physicians' Motor Club of Philadelphia, a Past-President of the Philadelphia County Medical Society, a charter member of Thomas R. Patton Lodge, F. & A. M., a member of Melita Chapter, R. A. M., the Society of the Sons of St. George, the Artisans Order of Mutual Pro-

tection, and a Past-President of the Philadelphia Medical Examiners' Association.

He took a great interest in Church life and had been, before his death, for many years an Elder in the Bethany Presbyterian Church and a teacher in the Sunday School for nearly forty years. He was a life long friend of John Wanamaker.

Dr. Gadd is survived by his widow, who was Eva Veitch Wilson before her marriage in 1888, two daughters, Esther Gadd and Jennie Gadd Hopkins, and a son, Wesley Gadd, formerly a General Agent of the Philadelphia Life Insurance Company and Manager at Wilkes-Barre, Pa., for the Fidelity Mutual Life Insurance Company, and now a member of the faculty at Franklin & Marshall College, Lancaster, Pa. Also two grandchildren, Samuel Wesley Gadd, 2nd, and Eleanor Elizabeth Gadd.

Above everything else, Dr. Gadd was a loving husband and proud father.

Those of us who had the privilege of knowing Dr. Gadd all feel a deep and personal loss in his passing from our midst. Life to him was sweet but he was ready to go, calling the time his "crowning day". He often wondered where life would have taken him if he had not been turned down to enter the Central High School.

DR. SCADDING—Before proceeding with the scientific portion of our program, I wish to present Mr. Leighton McCarthy, President of the Canada Life Assurance Company, who, I'm sure, is anxious to welcome all our members and guests to Toronto.

MR. MCCARTHY—Mr. President, Doctors, Gentlemen: I felt quite elated when a friend, whom I did not know, welcomed me to this meeting this morning with, "How are you, Doctor? I hope you are well". I replied I was quite well, and very much flattered. I don't know whether I look like a doctor or not, but I nearly was a doctor. At one time I hoped to be one.

When our good and charming friend asked me to do what I am about to endeavor to do, I was most delighted. He told

me that I would be placed on the program between business and science. I told him that that was a position which I seem to have occupied most of my life, i. e., the middle part of a sandwich. I am about to give you a very moderate sandwich. There won't be much meat in it.

It is my great pleasure to welcome this Convention and the members of it to the City of Toronto and to Canada. You have done Canada an honor in meeting here. A great feeling of pleasure passed over me when I learned that you had elected my good friend and colleague as your President for this year and that he was in consequence able to entice you to this city. I am glad he succeeded in bringing you this year. Perhaps next year it would have been more difficult. You will be able to give your own prescriptions without let or hindrance in the United States by this time next year, whereas you could not do it this year. But he can give you prescriptions without limit here and I hope he has done it bountifully, and that none of you are the worse for it.

It would be idle for me to tell you anything about your own Association. It is one that is greatly valued in the insurance world.

I have also had a very great respect for the medical profession. In fact, I had to have it; it was taught me from the cradle and if I didn't show that respect to one member, he was very expert with the strap. That was my father, who was a doctor.

I think, perhaps, by reason of the lessons he taught me and the respect he imbued in me, I have been able to treat with incredulity the allegation I frequently hear that the mortality experience in the non-medicals is better than in the medicals.

I don't propose this morning to try and tell you what a valuable Association you are. You know it better than I do. But as the Chief Executive of a company, I would like to say there is one thing, perhaps, above all, that we appreciate and that is the splendid and arduous work accomplished by this Association, together with the Actuarial Association, in what is called the "Joint Committee on Mortality". The volume of work is colossal. The results indicate what you have been able to do in the way

of standardizing the selection of risks. The practice and method of business introduced as applied both to the standard and the substandard business has been invaluable to the life insurance companies. They appreciate it more than any word of mine is capable of expressing.

I hope you will enjoy your visit to Toronto. I hope that it will be profitable, both from the business end and from the scientific end. I hope that it will be still more enjoyable in the other end of the business. I was playing behind some of your members on the golf course late yesterday afternoon. I hope that they succeeded in enjoying themselves. I believe in recreation as well as in conventions.

Without further ado, may I, on behalf of the life insurance companies and, in fact, the people of Canada, extend greetings and as warm a welcome as it is possible for this Country to give you?

DR. SCADDING—I know we're all delighted to have with us Dr. John R. Neal, Medical Director of the Abraham Lincoln Life Insurance Company and Chairman of the Medical Section of the American Life Convention, who has promised to say a few words to us.

DR. NEAL—Dr. Scadding has very graciously extended to the Medical Section of the American Life Convention an invitation to be represented in a few brief words on the program. No programs have contributed so richly to the better understanding and the proper procedure in the valuation of those who apply for life insurance and that, too, is the aim of the Section of which I have the honor of being the Chairman.

In looking over the program that will be presented to you, it is quite evident that you will add another chapter to your very enviable record. So, Mr. President, it is my very pleasureable duty and privilege to bring to this Association the greetings and best wishes from the American Life Convention and we trust that you will have a most successful meeting.

DR. SCADDING—To attempt to add anything to Mr. McCarthy's welcome would be to paint the lily. Let me simply say that I heartily endorse his sentiments.

You have done Canada and the Canada Life a distinct honor in coming here, and I trust you will not find us lacking in hospitality. For graciously electing me as your President, I ask you to accept my warm thanks and to believe that I and my fellow officers of the Association are your willing servants. There are certain famous men that I feel we all should praise. These are they who had the courage and sagacity to jettison the traditional presidential address. My only possible claim to fame is to follow in their train.

Whatever we have been doing in the past and may still be doing, I am persuaded we can do it better for the future.

If the business is non-medical, let us stick to principles that experience has taught us make for safety. Common honesty of the communities in this gigantic part of the world, while there are many notable exceptions, is rare. Dependable representatives, like dependable medical examiners, do not grow in every field. We should encourage our lay reporters and inspectors to dig deeper. In many instances they are not earning the paltry pittance we pay them.

If it is medically examined business, might I suggest weeding out the sports and weaklings from the perennial garden of medical examiners and fertilizing dependable specimens more liberally with argentum. No wonder prospective or actual applicants for insurance think we do not know our business when an individual, who is applying to say three companies at the same time, is declined by one, rated up by another, while still a third accepts the application on a standard basis. The remedy is some such guide as the Medical Impairment Ratings.

How unfair it is for some companies, when another, which is co-interested in a claim for Total Disability Benefit, goes off at "half cock" and, for any reason, I care not what, admits on grounds which might not be considered sufficiently weighty by others interested. There are no companies that I know of whose skirts are immaculately clean in this respect. There is

only one company that I feel at liberty to say has been guilty, my own. The remedy lies in hastening slowly in admission of all Total Disability claims of the doubtful and contentious varieties, and a thorough-going, whole-hearted cooperation of all companies interested. As Mr. Barney recently said at the meeting of the Medical Section of the American Life Convention, "We must stick together or 'get stuck' separately". This probably applies to selection of risks as well as admission of claims for Total Disability Benefit. Curses be upon the sirens that lured "the angel of life insurance" into adorning herself with such tawdry and expensive frills as Total Disability and Double Indemnity Benefits.

The first contribution to the program is by one of our distinguished guests, Dr. Israel M. Rabinowitch. Like most men who have climbed to the top, he had many obstacles to surmount in his early medical days—the victim of typhoid during his war training when he was Lance Corporal of the Canadian Army Medical Corps at Valcartier—a struggle against financial odds to acquire and equip a bio-chemical laboratory, on which his mind and heart were set. While, in the early days of his career, he was attached to the Montreal General Hospital, it had at that time no such laboratory. He was given a bare room in the Pathological Department with permission to work out his own salvation. Very soon he was producing original work of a very high order and with that came financial assistance and official recognition, the establishment of a Department of Metabolism, and his appointment as Chief. He is and has been an indomitable worker and diligent seeker after truth, and he is, as probably most of you know, a prolific contributor to medical literature. I am sure all of us who have read the paper he is about to present are astonished at the amount of statistical investigation it has obviously entailed.

Last year McGill University conferred upon him the degree of Doctor of Science, *honoris causa*, a distinction reserved for only those who have done outstanding work. He is Assistant Professor of Medicine and as outstanding a physician as he is a laboratory man—a rare but ideal combination.

THE DIAGNOSIS OF DIABETES

By I. M. RABINOWITCH

Statistics has its fallacies and diabetes affords no exception to the rule. Since the beginning of the present century, though the death rate from all causes has decreased approximately 45 per cent. in the total registration area of the United States, that from diabetes mellitus shows an increase of over 200 per cent.; in 1900, there were 1,780 deaths from all causes per 100,000 estimated population, compared with 980 in 1931—the latest available data; whereas, the death rate from diabetes increased from 9.3 to 20.4 per 100,000 estimated population during the same period.

The limited significance which must be attached to these findings is clearly shown when we study mortality by decades from 1880 to the present time. The data (see Table 1) indicate such

TABLE 1

Showing Deaths from all Causes and from Diabetes Mellitus per
100,000 Estimated Population in the Total Registration
Area of the United States.

| | 1880 | 1890 | 1900 | 1910 | 1922* | 1931 |
|------------------------------------|------|------|------|------|-------|------|
| Deaths from all causes | 1880 | 1960 | 1780 | 1500 | 1180 | 960 |
| Deaths from diabetes mellitus..... | 2.8 | 5.5 | 9.3 | 14.9 | 18.4 | 20.4 |

*1922 taken in place of 1920, as it separates the insulin from the non-insulin era.

rapid rise of mortality from diabetes that, if the rate continues to increase as rapidly during the next 50 years as it has during the last 50 years, it would not only exceed the combined mortalities from malignancy, tuberculosis, heart and kidney disease, but would account for practically all deaths. This, alone, is evidence that the increase in mortality is to some extent fallacious, when consideration is given to the fact that diabetics represent approximately 2 per cent. only of the population, and about 2 per cent. only of this group die annually. The general publicity given to the disease; more accurate methods of diagnosis and

TABLE 2
Showing Deaths from Diabetes Mellitus per 100,000 Estimated Population Before and Since Insulin.

| | 1921 | 1922 | 1923 | 1924 | 1925 | 1926 | 1927 | 1928 | 1929 | 1930 | 1931 | 1932 |
|---|------|------|------|------|------|------|------|------|------|------|------|------|
| England and Wales | 10.8 | 11.9 | 11.4 | 10.9 | 11.2 | 11.5 | 12.6 | 13.1 | 14.2 | 14.2 | 14.5 | 15.2 |
| London | 9.3 | 10.6 | 9.6 | 9.5 | 9.0 | 10.1 | 10.6 | 11.2 | 13.1 | 12.3 | 12.9 | 13.0 |
| Edinburgh | 10.5 | 12.3 | 10.6 | 11.3 | 10.2 | 10.6 | 9.0 | 9.0 | 14.0 | 13.0 | 13.9 | 10.9 |
| United States | | | | | | | | | | | | |
| Total registration | 16.8 | 18.4 | 17.9 | 16.6 | 16.9 | 18.0 | 17.5 | 19.0 | 18.8 | 19.0 | 20.4 | |
| Cities in registration States | 20.2 | 22.4 | 21.9 | 20.2 | 21.1 | 22.6 | 22.4 | 24.6 | 24.2 | 23.7 | 20.4 | |
| Rural part of registration States | 13.6 | 14.9 | 14.3 | 13.4 | 13.1 | 13.9 | 13.3 | 14.4 | 14.2 | 14.7 | | |
| Reg. Cities in non-reg. States | 17.0 | 15.1 | 14.9 | 12.3 | 15.3 | 14.7 | 14.1 | 16.6 | 16.2 | 15.0 | 14.8 | |
| All Registration Cities | 20.1 | 22.2 | 21.7 | 20.0 | 20.9 | 22.3 | 22.1 | 24.4 | 24.0 | 23.5 | | |
| Baltimore | 22.1 | 26.0 | 24.2 | 23.1 | 24.9 | 27.2 | 23.6 | 28.6 | 23.1 | 27.5 | 29.5 | |
| Boston | 19.8 | 29.3 | 24.8 | 23.7 | 20.9 | 25.8 | 23.7 | 23.2 | 30.9 | 30.7 | 30.2 | |
| Chicago | 20.3 | 23.2 | 21.8 | 18.8 | 21.5 | 25.6 | 22.5 | 24.4 | 25.0 | 23.4 | 27.1 | |
| New York | 24.1 | 27.9 | 27.4 | 25.7 | 25.9 | 27.7 | 26.5 | 26.6 | 26.5 | 26.7 | 29.7 | |
| Philadelphia | 19.1 | 20.5 | 20.9 | 21.3 | 21.9 | 23.6 | 22.4 | 27.1 | 26.1 | 25.0 | 29.9 | |
| St. Louis | 21.1 | 23.4 | 22.0 | 23.0 | 21.8 | 22.5 | 22.6 | 24.5 | 32.3 | 29.8 | 33.6 | |
| Canada | | | | | | | | | | | | |
| Ontario | 11.4 | 12.4 | 13.1 | 10.8 | 10.9 | 12.7 | 13.9 | 13.0 | 13.7 | 14.5 | 14.4 | 17.1 |
| Quebec | 7.8 | 8.3 | 7.8 | 9.0 | 8.1 | 10.0 | 11.0 | 4.0 | 12.3 | 11.2 | 13.0 | 12.2 |
| Toronto | 12.6 | 14.5 | 15.6 | 13.1 | 12.8 | 15.6 | 15.1 | 15.0 | 16.0 | 15.9 | 17.9 | 20.4 |
| Montreal | 11.0 | 10.9 | 10.7 | 13.1 | 10.7 | 13.7 | 14.1 | 13.6 | 16.9 | 11.3 | 18.8 | 20.8 |

increased attention paid to the disease by the physician (routine urinalysis) account largely for the apparent increase in mortality.

During the last ten years, the death rate from diabetes mellitus has remained remarkably stationary, in spite of improved methods of treatment. This is shown in Table 2. It would, therefore, appear that more exact methods of diagnosis and routine urinalysis are now operating with full force and effect; that the influence that they can exert is at a maximum; or that apparent increase of mortality due to more exact reporting of deaths, is probably counter-balanced to some extent at least by a decrease due to improved methods of treatment. The result is a stationary death rate.

This stationary death rate has one advantage from the point of view of Life Assurance work. Experience in industry in general may be cited as an analogy. Manufacturers are not as much concerned with the height of the cost of materials as they are with fluctuation of prices; high cost is of little moment, providing the manufacturer knows it will remain stationary. Unless I misunderstand the problem, the chief difficulty of Life Assurance Companies in determination of hazard is not the height of death rate (providing it is known) but, fluctuation of death rate. Fluctuation defies forecasting the cost of insurance.

Stationary as the present death rate from diabetes mellitus may appear to be, compared with the recent past, it is still high and very little comfort is found in the fact that, when mortalities are related to age, the increased rates are largely due to deaths amongst adults. Life assurance companies are largely concerned with adults, not with children amongst whom diabetes contributes little to mortality. (Approximately five per cent. of all diabetics are children.)

That these data do not reflect the progress made in the treatment of diabetes is clearly shown by comparison with hospital statistics. Experience with tuberculosis, surgical conditions and coma, complications and sequelæ of diabetes, may be cited as examples. A little over a generation ago tuberculosis was common and almost every second death amongst diabetics was due to it; whereas, now tuberculosis is probably no more common

than amongst non-diabetics. This statement is based upon the combined statistics of Adams, Fitz, Joslin and our own clinic. These show that amongst 8,029 diabetics, active pulmonary tuberculosis was found in 129 cases only—an incidence of approximately 1.25 per cent.

Surgery accounted for many deaths amongst diabetics in the past. In The Montreal General Hospital alone, over 700 diabetics were operated upon during the last ten years; and the mortality was no greater than amongst non-diabetics, when due consideration is given to age, general condition and type of operation. With proper pre-operative and post operative care, diabetes is no longer a contra-indication to surgery.

Coma is rapidly disappearing. In our clinic alone, though the total number of diabetics admitted between July, 1932, and July, 1933, was greater than during the corresponding period of the previous year, we had one case only of coma. This, in my opinion, is largely a reflection of the education of the average physician. Diabetes is now more often detected in its early stages because of routine urinalysis. Also, because of the publicity given to the disease, diabetics are more familiar with the signs and symptoms of approaching coma and with the conditions which may lead to it. The fact that we have had one case only in twelve months may have been an accident. It, nevertheless, represents the experience of a large clinic and in which the annual number of admissions for coma is decreasing. It at least shows that this condition is rapidly disappearing as a cause of death.

Aside from the conditions which I have mentioned, the future of the diabetic was not very bright a decade ago. Fortunately, then, as now, in the majority of cases the diabetes was mild, but the outlook of the severe diabetic was tragic. If he failed to follow treatment he was certain to die of coma; if he followed treatment he was almost equally certain to die of starvation. With better dietetic management and insulin, conditions changed fundamentally; the ratio of actual to expected deaths amongst carefully treated diabetics is, at present, not much greater than that of non-diabetics. This is shown in the following Table.

The Diagnosis of Diabetes

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Percentage Ratios of Actual to Expected Deaths (All Causes) in the Clinic for Diabetes at the Montreal General Hospital.

| | | | |
|------------|-----|------------|-----|
| 1922 - - - | 660 | 1927 - - - | 107 |
| 1923 - - - | 280 | 1928 - - - | 124 |
| 1924 - - - | 243 | 1929 - - - | 112 |
| 1925 - - - | 200 | 1930 - - - | 118 |
| 1926 - - - | 109 | 1931 - - - | 111 |
| 1932 - - - | 112 | | |

It will be observed that, whereas, in 1922, the ratio was 660 per cent. of the normal, in 1932 it was 112 per cent. only.

With respect to the above tabulation, it should be particularly noted that I used Glover's Life Table—Original Registration States (U.S.A.)—1910 Both Sexes, which made the expected deaths considerably higher than if I had used a table more representative of life insurance mortalities, such as the Canadian Men or American Men, and that all deaths from whatever cause were included (as it happened, there were two deaths only from violence). Since the material in the group is homogeneous with respect to one disease only, namely, diabetes—in other words since it is not a select group, all manner of pathological conditions being included,—it follows that the death rate in excess of the normal, by the table used, is only partly attributable to diabetes. Just what proportion of the excess mortality is directly and indirectly attributable to diabetes, I am unable to say, having made no attempt to classify the deaths into those of diabetic origin and non-diabetic origin. Stated otherwise, the relative mortalities as shown must be considerably higher than would have been the case, had the group consisted entirely of persons with no other obvious abnormality than that of being diabetic. It appears to me that you, gentlemen, will regard this method of analysis as satisfactory from the life insurance standpoint.

If Life Assurance Companies were to accept diabetics as policyholders on the mortality experience of the clinic at The Montreal General Hospital in 1932 (112 per cent.) and were to follow the practice of demanding ratings to cover twice the extra mortality actually experienced in 1932 (124 per cent.), the ratings to be demanded would, I believe, be approximately three and

one-half years. But Life Assurance Companies will not accept those who are frankly diabetic, regardless of the experience quoted, for the simple and sufficient reason that hospital statistics are of little or no practical value in life assurance. This is to be accounted for by two reasons: firstly, they do not represent conditions obtained amongst diabetics in general and, secondly, their number is small.

Life Assurance Companies will not only not accept diabetics, at least not at standard rates, but try to avoid or penalize *potential* diabetics, by making use of every available and practical procedure for detecting the disease. Blood sugar studies represent one of the more recent methods and I propose to deal largely with this phase of diagnosis of diabetes.

A decade ago, in using laboratory procedure for estimating risk, examiners of applicants for life assurance policies had to be satisfied with urinalysis only and a variety of methods were employed; urine was examined in the fasting state, after an ordinary meal, and after a rich carbohydrate diet; there were morning and evening samples; examination on four or five successive days; and an attempt was made to relate risk to frequency of glycosuria. With the development of practicable methods for estimation of blood sugar, the problem has become no less complicated. The laboratory records of The Montreal General Hospital at least show a variety of requests from the companies. They include examination of blood obtained in the fasting state and at various intervals after various meals. Nor does there appear to be any uniformity in the use or interpretation of the blood sugar time curve. One type of request includes examination of the blood in the fasting state and two hours after administration of 100 grams of glucose; another includes a fasting blood sugar and one and two hours after glucose; another also requires examination one-half hour after glucose, and some relate the amount of glucose to be given to body weight. In some cases there is a specific request that the test is to commence in the fasting state, while other companies appear to be satisfied with any time of the day, providing some hours have elapsed after

the last meal. Very few companies request a description of the blood sugar method used and some permit micro-chemical procedures, though we do not use them in our laboratory.

When due consideration is given to the many variables in the interpretation of blood sugar, I do not hesitate to suggest that life assurance companies have probably been misled more often than guided by this test, because of lack of uniformity; and unless the test is performed under proper conditions it would perhaps be as well to confine examination to urinalysis alone for, as I believe I shall demonstrate, urinalysis still has a definite place in evaluation of risk from diabetes. Glycosuria still implies diabetes in the vast majority of cases; renal glycosuria and pentosuria are comparatively rare.

The observations which I propose to make, particularly with regard to blood sugar time curves, must be accepted with some reservations as they represent the experience of one clinic only. I should, however, like to point out that they represent ten years of experience with more than 5,000 curves obtained under my supervision and under the same conditions with respect to preparation of patient, type of sugar, amount of sugar, and chemical technique. They include uniform histories, careful correlation of clinical with laboratory data and follow-up records in 2,280 individuals.

As stated, a single estimation of blood sugar in the fasting state is not an uncommon request. The value of such examination may be summarized very briefly. Unless the blood sugar is found increased, it is of little or no value from the point of view of determining insurability. Hyperglycæmia in the fasting state is seldom seen except in fairly advanced diabetes. In the mild diabetic, the blood sugar is usually normal in the fasting state, unless the disease is complicated by some condition which, per se, may cause hyperglycæmia (infection, hyperthyroidism, etc.) In the majority of cases the diabetes is mild, and it is this type of individual who is most frequently met with in life assurance work. Parenthetically, it may here be observed that the blood sugar is also usually normal in the fasting state in the

advanced diabetic, provided the disease is under control. A normal fasting blood sugar, therefore, does not exclude diabetes. But as I propose to show, single estimation of blood sugar has its value.

Assurance companies usually request blood sugar time curves when there is a history of glycosuria or some other sign suggestive of diabetes. Therefore, when, in a single examination, the blood sugar is found increased, it indicates disturbed carbohydrate metabolism, with extremely few exceptions. Therefore, such examinations may save the individual the inconvenience and expense of a blood sugar time curve, a matter of importance to those companies who do not accept sub-standard risks. The reliability of hyperglycemia in the fasting state is shown in the following Table from one of my earlier publications:

Relationship Between Fasting Blood Sugar and Type
(Positive or Negative) of Blood Sugar Time Curve.

| Fasting Blood Sugars (Per cent.) | Number of Cases | Diminished COH Tolerance | |
|--|--------------------|--------------------------|-------------------------|
| | | Number of Cases | Percentage Incidence |
| 0.121-0.125 | 23 | 14 | 60.8 |
| 0.126-0.130 | 23 | 18 | 78.2 |
| 0.131-0.135 | 9 | 8 | 88.2 |
| 0.136-0.140 | 14 | 13 | 92.8 |
| 0.141-0.145 | 7 | 7 | 100.0 |
| 0.146-0.150 | 6 | 6 | 100.0 |
| 0.151-+ | 21 | 21 | 100.0 |

It will be observed that when the fasting state sugar was 0.14 per cent. or more, all of the blood sugar time curves indicated disturbance of carbohydrate metabolism. I first reported these results in 1926 (*J. Clin. Invest.*, vol. 2, p. 579, 1926). Since, at that time, the number of observations was small, limited significance could be attached to the percentages. But corroboration of the results came from subsequent analyses made upon 1,346 individuals, which I reproduce below (Rabinowitch, *Diabetes Mellitus*, 1933, Macmillan and Company, Limited).

Relationship Between Fasting Blood Sugar and Type
(Positive or Negative) of Blood Sugar Time Curve.

| Fasting Blood Sugars (Per cent.) | Number of Cases | Diminished COH Tolerance | |
|--|--------------------|--------------------------|-------------------------|
| | | Number of Cases | Percentage Incidence |
| 0.121-0.125 | 421 | 272 | 64.6 |
| 0.126-0.130 | 236 | 191 | 80.9 |
| 0.131-0.135 | 198 | 173 | 87.3 |
| 0.136-0.140 | 125 | 121 | 96.8 |
| 0.141-+ | 366 | 365 | 100.0 |

From these it will, again, be observed that a blood sugar of 0.14 per cent. or over, when found in the fasting state, definitely indicates disturbance of carbohydrate metabolism. In such cases blood sugar time curves are not necessary (unless with a view to sub-standard insurance).

A blood sugar time curve is, however, indicated in every suspect when the fasting blood sugar is normal. It is the most sensitive test presently available for the detection of disturbance of carbohydrate metabolism. It is the first indication of diabetes to appear and the last to disappear. To apply this test intelligently in practice, however, it is important to recognize the many variables which may influence the results.

Many of the remarks which I shall make may be familiar to some of this audience and in places appear elementary. May I, however, observe that these men are teachers and no teacher finds it uninteresting, especially in moments of leisure, to return to fundamentals. Repetition, so far as I know, has never as yet done harm.

What is a blood sugar time curve? This test is more commonly, but erroneously, known as a "sugar tolerance test". It is a series of measures of the sugar content of the blood in relationship to time elapsed following administration of a glucose meal; and is essentially a measure of the disproportion between the rate of absorption of glucose from the gastro-intestinal tract and the rate of its utilization by the tissues—utilization implying the combined mechanisms of oxidation and storage. Hyperglycemia results when the rate of utilization cannot keep pace

with the rate of absorption. Increased glycaemia is a normal response to ingestion of glucose. It is, however, *transitory* and the rate at which the blood sugar returns to the normal level is, in general, an indication of the efficiency of the carbohydrate metabolism of the individual. In diabetes, either because of defective storage or defective oxidation, the rate at which sugar is removed from the blood cannot keep pace with the rate at which it enters. The result is not transitory, but *prolonged*, hyperglycaemia.

A complete curve includes examination of the blood in the fasting state and again at well chosen intervals (30, 60, 120 and 150 minutes) after administration of glucose; and has the following attributes under normal conditions:—In the fasting state the blood sugar ranges between 0.08 and 0.12 per cent. After ingestion of glucose, the maximum hyperglycaemic response is usually noted at the 30 minute period and the blood sugar is then below 0.18 per cent., ranging between 0.15 and 0.17 per cent. At the two hour period, not only does the blood sugar return to its original level, but drops below it. (The usual explanation of this is stimulation of glycogenesis.) At the two and one-half hour period, the blood sugar is at its original level.

With disturbance of carbohydrate metabolism, the peak of the curve is more marked and usually develops more slowly, the blood sugar reaching its maximum level in an hour or more, instead of thirty minutes, after glucose ingestion. The curve of descent, that is, the time it takes for the blood sugar to return to the original level, is prolonged and, at some time during the test, sugar appears in the urine.

As stated, a number of factors influence the curve; and their recognition is essential for proper interpretation of data. Failure to perform the test under proper conditions may account for a normal curve in a diabetic, and a diabetic curve in a normal, individual. I shall refer to these conditions briefly.

The first, and most important, condition is that there must be no alteration whatever of the dietary habits of the individual prior to the test. This was very clearly demonstrated by Nielson (Bioch. J., vol. 22, p. 1490, 1928). Prior restriction of carbo-

hydrate intake may account for a perfectly normal curve in a mild diabetic, but, in our experience at least, it is the rule, rather than the exception, that diets are altered prior to the test. The usual experience is that sugar is discovered during an examination for life assurance. The individual then applies to his physician for advice and restriction of carbohydrates is suggested. Shortly after, still anxious to obtain a policy, he again applies and a blood sugar time curve is requested.

There is one advantage to the companies if an applicant resorts to starvation prior to the test. If continued for some time, carbohydrate tolerance is disturbed and the diabetic response is more pronounced and more readily detectable. The condition then somewhat resembles that produced by prolonged starvation. Carbohydrate tolerance may be so disturbed by restriction of carbohydrates that insulin may not be helpful. (J. Biol. Chem., vol. 100, p. 493, 1933.) For normal carbohydrate metabolism, daily stimulation of the insulin-producing mechanism by carbohydrates is essential. (J. Biol. Chem., vol. 83, p. 747, 1929.)

The effects of sudden restriction of carbohydrates are at times most striking. Acetone may appear in the urine within twenty-four hours; and though the blood sugar may be normal or actually reduced in the fasting state, the blood sugar time curve may be typically diabetic; the peak develops slowly and reaches a high level and the time taken for the blood sugar to return to the normal level is prolonged. Theoretically, one might expect the opposite; since restriction of carbohydrates depletes the storage of glycogen, one would expect rapid disappearance of glucose from the blood, following its ingestion. Apparently, the ketonæmia—the acetone in the blood—impairs the glycogenic function of the liver. In my experience, at least, the development of acetone invariably precedes the disturbance of the blood sugar curve. A practical application of this observation is that reports of blood sugar times curves should be accompanied by reports of acetone in the urine. If the blood sugar time curve is normal and the urine contains acetone, one should suspect some change of diet prior to the test.

The test should be performed in the morning and in the fasting state. This choice of conditions is not entirely arbitrary. It is based upon a variety of experiences, the majority of which favour the morning.

In properly conducted experiments, the peak of the curve is usually higher in the afternoon. Since excretion of glucose is dependent upon a renal threshold, glycosuria is more likely to occur with a high than with a low peak. For this reason, it would appear that diabetes is more readily detected in the afternoon.

Glycosuria is also more likely to occur in the afternoon because of variation of the renal threshold for glucose. That there are at least two thresholds for glucose when the blood sugar is falling has been recognized for some time^(1,2). More recently it has been shown⁽³⁾ that the threshold for the rising blood sugar may change during the day; it may be higher in the morning than in the afternoon and vice versa. In properly conducted experiments, it is usually higher in the afternoon.

Though the peak and the renal threshold favour afternoon examination, diabetes is more likely to be overlooked than detected by the blood sugar time curve at this time of day, because of a number of other conditions, chief of which are the effects of food and muscular activity.

Administration of glucose after an ordinary meal for detection of diabetes was apparently originated by Naunyn. The practice was to give the patient a breakfast of 80 to 100 grams of bread and a cup of coffee with milk. Two hours later, he was given 100 grams of glucose. If appreciable glycosuria appeared, it was regarded as evidence of disturbed carbohydrate tolerance. The possibility of stimulation of glycogenesis by carbohydrates was then not recognized. It is now known that a carbohydrate-rich diet may so stimulate insulin production as to account for a normal curve in a diabetic, providing the disease is mild and a few hours only have elapsed since the ingestion of food.

1. Frank, *Arch. exp. Path. Phar.*, vol. 72, p. 387; 1913.

2. Rabinowitch, *J. C. M. A.*, vol. 83, p. 329; 1930.

3. Harding and Selby, *Bioch. J.*, vol. 26, p. 957; 1932.

Stimulation of glycogenesis (storage of sugar as glycogen) by carbohydrates is readily demonstrated in normal individuals by obtaining a second curve with another 100 grams of glucose *immediately* after the first test is completed. The following is an example:

| FIRST CURVE | | SECOND CURVE | |
|------------------------|-------------|------------------------|-------------|
| Time | Blood Sugar | Time | Blood Sugar |
| Before glucose | 0.103 | | |
| 30 minutes after | 0.178 | 30 minutes later | 0.143 |
| 60 " " | 0.143 | 60 " " | 0.120 |
| 120 " " | 0.087 | 120 " " | 0.095 |
| 150 " " | 0.111 | 150 " " | 0.103 |

It will be observed that the peak of the curve was definitely lower after the second dose of glucose.

As a rule, when tests are to be done in the afternoon, it is specified that they are to commence some hours after the last meal. This practice, however, may result in irregularities of the curve, largely due to variations in digestion and absorption of glucose-producing food materials.

Muscular activity is an important variable. It leads to greater utilization of carbohydrates—a fact made use of in the treatment of diabetes. When not taken to the point of fatigue, exercise enables the severe diabetic to decrease his doses of insulin; and for the same reason may account for a perfectly normal curve in a mild diabetic. This was very forcibly brought to my attention in a case in which a curve was obtained following a strenuous game of golf on the previous day. Obviously, the effect of muscular activity is more or less automatically eliminated when blood sugar time curves are obtained in the morning before breakfast instead of later in the day.

In view of the possible effects of exercise, it is obvious that the individual should be at rest during the entire period of the test, if slight defects of carbohydrate metabolism are to be detected.

Aside from the conditions I have mentioned, a not unimportant factor is comparability of data. The generally accepted standards are based upon the results of tests performed in the morning.

It, therefore, appears logical to adhere to this practice, in order to compare things with things that are comparable.

Irregular curves are frequently due to inaccurate timing of collection of blood. To give the subject a drink of glucose and return at the stated intervals for the collection of blood regardless of the time the drink was finished may account for almost any type of curve. In this Clinic, it is the practice to allow the patient five minutes only for the drink. The time is then taken and recorded and blood is collected exactly 30, 60, 120 and 150 minutes later.

Clumsy insertion of a needle during the venous puncture or the use of a blunt needle may occasionally cause hyperglycemia. The phenomenon is somewhat similar to that following the injection of adrenalin.

The drink must be palatable. As stated, this test is essentially a measurement of the difference between the rate of absorption of glucose from the alimentary canal and the rate at which the absorbed glucose is utilized. Hyperglycemia results when the combined rates of storage and oxidation cannot keep pace with the rate of absorption. On the other hand, if the mechanism of absorption is impaired, the rate at which the ingested glucose reaches the tissues may be so slow that hyperglycemia may not be produced, in spite of some defect of storage or oxidation. Under these conditions, it is possible to obtain—and I now quote our own experiences—normal curves in individuals definitely known to be diabetic. Excluding organic disease of the gastrointestinal tract, a common cause of defective absorption of glucose is faulty preparation of the drink. In view of the importance of this part of the test, a few observations will be made with regard to it.

A common practice is to give 100 grams of glucose dissolved in a glassful of water flavoured with lemon juice. The solution is, therefore, hypertonic. In the mixture used by us, the total volume of liquid is 250 cubic centimeters. This represents a 40 per cent. solution of glucose. Hypertonicity may, per se, be responsible for defective absorption of glucose from the gastrointestinal tract. In our experience, however, the greatest source

of error lies in the ease with which it is possible to produce a most unpalatable and nauseating drink, unless careful attention is paid to the flavouring and chilling. With an unpalatable or nauseating drink not only may the curve be normal, but perfectly *flat*—suggesting no absorption whatever of glucose from the alimentary tract. Flat curves may, of course, be due to hyperinsulinism. Life assurance companies are, however, not much concerned with this phenomenon because of its rarity. From the point of view of insurability of the individual, a flat curve should indicate repetition of the test; it should not be assumed that there is no disturbance of carbohydrate metabolism.

When properly flavoured and chilled, a glucose drink can be made very attractive*, especially when given after the twelve to fifteen hour fasting period necessary before the test. In this hospital, no drinks are prepared in the wards; all are prepared in the metabolism laboratories; they are kept there on ice until required and are tasted before they are given to patients. Incidentally, at each blood collection, careful note is made of nausea and any possible mishap during the test (loss of urine, accidental ingestion of other food or fluids, etc.)

Amount of Sugar Used in Test—The amount of sugar used in the test is important in that it governs, in general, the rate of decay of the curve, that is, the rate at which the blood sugar returns to the normal level. It does not appreciably influence the peak of the curve; ten grams of glucose may raise the blood sugar as much as 50 grams. As the majority of workers agree that the time the blood sugar returns to the normal level is more significant than the "peak" of the curve, it is important that the individual be given a *sufficient amount* of glucose.

Methods of Estimating Blood Sugar—Though there are a variety of methods of estimating blood sugar, in principle, they have much in common. The first procedure usually consists of laking the blood and precipitation of proteins, in order to obtain a clear solution after filtration. A known amount of the clear filtrate is then treated with some reagent capable of yielding up

*Some idea of how attractive this drink can be made may be gained from the fact that patients suffering from acute febrile disorders (pneumonia, etc.) may be induced to take five to six hundred grams of glucose a day.

the oxygen necessary for oxidation of the aldehyde group of the sugar molecule. The degree of reduction of the reagent is then measured and taken as an index of the amount of sugar present. In general, the procedures fall into two groups, namely, titrimetric and colorimetric. The former include such methods as the Bang, Maclean, Hagedorn-Jensen and Shaffer-Hartmann. The variety of Folin procedures are colorimetric and depend upon the reduction of an alkaline copper solution with the formation of cuprous oxide and estimation of the latter by its oxidation with another reagent, which in turn is reduced with formation of a blue colour, the intensity of which is proportional to the amount of cuprous oxide, which, in turn, is proportional to the amount of sugar present.

With regard to results in general, colorimetric methods yield less exact values than titrimetric. Though some approach specificity, no one test is absolutely specific for glucose. In selection of a method from those mentioned, there are, however, a number of considerations other than specificity; and, the most important, from the practical point of view, is *comparability of data* and this postulates consistent use of the same or very similar methods. Special precautions are necessary with the use of more recent methods. They are more specific for glucose and, therefore, yield lower values than the older methods; according to the latter, the normal fasting blood sugar ranges between 0.08 and 0.12 per cent.; whereas, with the newer methods, it ranges between 0.05 and 0.08 per cent. only. By the use of newer methods and older standards of normality, it is obvious that slight disturbances of carbohydrate metabolism may be readily overlooked.

Though there may be little to choose between the different macro-chemical procedures I have referred to, I should like to point out a possible fallacy in the use of micro-chemical technique with capillary blood. I am not suggesting that these micro-chemical methods cannot be exact. The values obtained with them correspond to the macro procedures when careful attention is paid to *all* details. As will be seen presently, an appreciable source of error lies in the collection of the blood.

Capillary blood is largely a mixture of arterial and venous blood and, depending upon conditions during collection, the blood sugar may be made to correspond to practically pure arterial, pure venous, or a mixture of both arterial and venous blood. When there is no interference with the flow of blood and when the finger is kept in warm water for some time before puncture, the blood sugar corresponds closely to that found in the arteries. On the other hand, if the skin is cooled, or if there is interference with the flow of blood, the sugar tends to approach that found in the veins. Though the difference between arterial and venous blood sugar may be inappreciable in the fasting state, it may be quite marked after ingestion of glucose. This is shown in the following arterial and venous blood sugar time curves obtained simultaneously:

| | Fasting | 30 min. | 60 min. | 120 min. | 150 min. |
|------------------|---------|---------|---------|----------|----------|
| Arterial | 0.102 | 0.240 | 0.181 | 0.140 | 0.123 |
| Venous | 0.100 | 0.168 | 0.129 | 0.108 | 0.096 |
| Difference | 0.002 | 0.072 | 0.052 | 0.032 | 0.027 |

It will be observed that though the values of arterial and venous blood are practically the same in the fasting state, one-half hour after the glucose was given the difference between the arterial and venous blood sugars was 72 mgms.; and not only was the peak of the curve higher with the arterial blood, but the curve of decay—the rate at which the blood sugar returned to the normal level—was slower. One possible error inherent in blood sugar time curves obtained with capillary blood is, therefore, obvious; and it is not entirely theoretical. I, here, reproduce a pair of curves from the same patient; the figures by the micro-method coming to me from Dr. C. C. Birchard of the Sun Life Assurance Company of Canada. This case is cited especially because both tests were performed in well organized hospitals; in well equipped laboratories; and by persons quite expert in blood sugar analyses. The error, in the micro figures, in my opinion, occurred during the collection of the blood sample.

On May 26th, 1933, a blood sugar time curve was obtained with capillary blood. One hundred grams of glucose were then given by mouth. The following were the results:

| | | | | | BLOOD SUGAR (per cent.) |
|--------------------------|---|-----|---|--|----------------------------|
| Fasting | | | | | 0.24 |
| At the end of 30 minutes | | | | | 0.28 |
| " | " | 60 | " | | 0.24 |
| " | " | 120 | " | | 0.23 |
| " | " | 150 | " | | 0.19 |

When I was shown this curve, I suggested to Dr. Birchard that it was due to diabetes, providing there was no other clinical condition (hyperthyroidism, etc.) to account for the hyperglycæmia. A rather unusual finding was the absence of any marked increase in glycæmia from the ingestion of glucose. In my experience at least, a fasting blood sugar of 0.24 per cent. is usually followed by a very high peak, the blood sugar reaching 0.40 or 0.50 per cent., or higher. I suggested that its absence in this case might have been due to defective absorption of sugar from the gastro-intestinal tract. Dr. Birchard then informed me that the man was perfectly well; there were no clinical signs nor symptoms to suggest diabetes and there was no history of glycosuria. I then suggested that the test be repeated using a macro procedure with ordinary venous blood. This was done four days later, using 3 c.c. quantities of blood obtained from the arm vein, with the following results:

| | | | | | BLOOD SUGAR (per cent.) |
|--------------------------|---|-----|---|--|----------------------------|
| Fasting | | | | | 0.114 |
| At the end of 30 minutes | | | | | 0.151 |
| " | " | 60 | " | | 0.137 |
| " | " | 120 | " | | 0.104 |
| " | " | 150 | " | | 0.091 |

There were no changes in the diet between the two tests.

Assuming uniformity of preparation for the test and uniformity of technique, there remain two attributes of the curve, the significance of which is, as yet, imperfectly understood. Though there is unanimity of opinion with respect to the characteristics of a *perfectly normal* and a definitely abnormal curve, there is doubt about the significance of a high peak (a maximum

blood sugar greater than 0.18 per cent.), and the rate of development of the peak. Many ignore the peak entirely, regardless of its height. For example, Graham of London (*Lancet*, Jan. 26th, 1929) regards a blood sugar as high as 0.23 per cent. at the 30 minute period as normal, providing the blood sugar is below 0.13 per cent. at the end of two hours. Some do not ignore the height of the peak. Extremely few pay attention to the rate of development of the peak, that is, whether the maximum hyperglycæmic response is noted 30 minutes after glucose ingestion or later.

Working with the same problem daily, one cannot help but be impressed with phenomena as they occur repeatedly and it has been my impression for some years that both attributes of the peak—height and rate of development—are essential for proper interpretation of data. Accordingly, these phenomena were investigated. Curves were correlated with carefully obtained clinical histories and physical findings.

This study included 2,280 individuals in many of whom curves were obtained periodically. Amongst those in whom tests were repeated, some were given treatment, while others were instructed to follow their ordinary dietary habits. They were, however, told that diabetes was suspected and that it would be advisable to have the tests repeated periodically. The data obtained, as I shall show, appear to support the view that the peak is important; that a blood sugar greater than 0.18 per cent. at any time after ingestion of glucose indicates abnormal carbohydrate metabolism in the majority of cases. The data also appear to indicate that the rate of development of the peak is important.

The first investigation was concerned with the family history of diabetes. Evidence is rapidly accumulating that diabetes is largely an hereditary disease, and it would appear that it is transmitted as a recessive unit character (*Ann. Int. Med.*, vol. 6, p. 1272, 1933; *Am. J. Med. Sc.*, vol. 186, p. 1, 1933). There is, however, much to suggest that it may at times be a dominant trait. The occurrence of diabetes in homologous twins is significant, as it is presumptive evidence of hereditary transmission. There are now about 20 such cases in the literature. (J. Mich.

State Med. Soc., June, 1933.) Naunyn made the observation that the more carefully one inquired into the family history of diabetes, the more commonly it is met with. Joslin, at one time, stated that he was also convinced of this, in spite of the fact that diabetics are more likely to know more of the presence of the disease in the family than those who are not diabetic. The importance of hereditary is fully appreciated only when family history is carefully gone into. When first obtained it may be negative, but later questioning may bring out the fact that some relative has died of diabetes or that some one has since developed it. The hereditary nature of diabetes is, at times, strikingly borne out by a child showing it before the parent or the grandparent. In such a case the probability is that parent or grandparent were potential diabetics, but the necessary conditions to make the disease active (infection, etc.) were absent.

As a control, in this investigation, 1,000 records of non-diabetics from the medical services of The Montreal General Hospital were analyzed. In an investigation such as this, uniformity and completeness of history-taking are obviously important. This, of course, applies to any medical investigation, but it does not appear to be generally recognized. From the standpoint of scientific record-taking, case histories, as Raymond Pearl has pointed out (*Medical Biometry and Statistics*, 1923, W. B. Saunders) are glaringly defective in what they *fail* to record about the patient. In this investigation, for example, records with the statement 'family history negative' were excluded. Reports were accepted as controls only when diabetes was specifically stated to have been present or absent. As an additional control, the records of 2,500 diabetics were similarly analyzed. Uniform histories are obtained in all cases of diabetes in this Clinic. The routine form which must be completed in every case contains the following with respect to family history:

Is there a family history of diabetes? If so, complete the following:

| Relationship | Age at onset | Present age | Age at death |
|--------------|--------------|-------------|--------------|
| | | | |

The family histories of the thousand non-diabetics and twenty-

five hundred diabetics were divided into (a) hereditary, and (b) familial—hereditary implying diabetes in a parent, grandparent, uncle, aunt or child, and familial implying diabetes in a sister, brother or cousin. Conjugal diabetes was not considered as significant. It was included amongst the negative findings, for there is reason to believe that this form of diabetes is not due to transmission of disease from husband to wife or vice versa, but to similarity of living. The combined data are shown in Table 3.

It will be observed that the *total* incidence of family history of diabetes—hereditary and familial—amongst the diabetics was three times greater than amongst the non-diabetics, and that the incidence amongst the individuals in whom blood sugar time curves were requested because disturbance of carbohydrate tolerance was suspected (Vide *infra* Table 4) was approximately equal to that of the diabetic group.

The data appear to be significant. As stated, the high incidence of family history of diabetes amongst diabetics is often explained by the fact that relatives of diabetics are more likely to know about the disease than relatives of non-diabetics. This does not explain the high incidence noted in this group of cases with blood sugar time curves, for the great majority of these individuals, being suspects only, were questioned for the first time with regard to family history of diabetes.

The findings are still more striking when the individuals are divided into two groups, namely, (a) those who applied for life assurance and in whom glycosuria was discovered previously, and (b) those in whom blood sugar time curves were requested for other reasons, but in the great majority of whom there was no glycosuria. The data are shown in Table 4.

It will be observed that the incidence of family history of diabetes was 18.8 per cent. in the non-insurance group; whereas, in the insurance group with glycosuria, it was 25.5 per cent.

These data, in my opinion, emphasize the diagnostic value of family history. I have the impression that applicants for Life Assurance are asked about *death* from diabetes in the family; but are not questioned as to whether some one in the family *has* the disease.

TABLE 3
FAMILY HISTORY OF DIABETES

| Subjects | Number | Negative | | Hereditary | | Familial | | Total Positive | |
|--------------------------------------|--------|----------|-----------|------------|-----------|----------|-----------|----------------|-----------|
| | | No. | Per cent. | No. | Per cent. | No. | Per cent. | No. | Per cent. |
| Non-diabetics | 1000 | 927 | 92.7 | 53 | 5.3 | 20 | 2.0 | 73 | 7.3 |
| Diabetics | 2500 | 1953 | 78.1 | 358 | 14.3 | 189 | 7.6 | 547 | 21.9 |
| Cases with blood sugar time curves.. | 2280 | 1825 | 80.0 | 326 | 14.4 | 129 | 5.6 | 455 | 20.0 |

TABLE 4
FAMILY HISTORY OF DIABETES

| Subjects | Number | Negative | | Hereditary | | Familial | | Total Positive | |
|---------------------------|--------|----------|-----------|------------|-----------|----------|-----------|----------------|-----------|
| | | No. | Per cent. | No. | Per cent. | No. | Per cent. | No. | Per cent. |
| Insurance group | 377 | 281 | 74.5 | 70 | 18.6 | 26 | 6.9 | 96 | 25.5 |
| Non-insurance group | 1903 | 1544 | 81.1 | 256 | 13.5 | 103 | 5.4 | 359 | 18.9 |

Because of the above findings, an attempt was made to determine the significance of the peak of the blood sugar time curve with the aid of family history. For this purpose, the above mentioned 2,280 cases were divided into groups depending upon the type of curve. Thus:

1. Negative
2. Positive
3. Negative except for high peak or slow rise
4. High peak alone
5. Slow rise alone

The combined data are shown in Table 5.

It will be noted that the total incidence of positive family history is high in every group, except for the negative curves (10.3 per cent.). Relative to the rate among normal controls (see Table 3) the percentage mentioned was also high, namely, 10.3 compared with 7.3 per cent. This may be due to the fact that some of these individuals are not normal with respect to carbohydrate metabolism; otherwise, they would not have been subjected to the test.

The data appear to be still more significant when the insurance cases with glycosuria are separated from the non-insurance group. The combined data are shown in Table 6.

It will be observed that, though the incidence of positive family history of diabetes is high in all groups, except for negative curves in the non-insurance cases, it is higher in the insurance cases with glycosuria, regardless of the type of curve. It is of interest to note that the incidence in the non-insurance group with the negative curves is approximately the same as in the normal controls (see Table 3).

If we accept it as a fact that family history is a reliable guide in the diagnosis of diabetes, the above data lead to the conclusion that the peak of the curve is of diagnostic importance; that a peak corresponding to a blood sugar of greater than 0.18 per cent. indicates disturbance of carbohydrate metabolism in the great majority of cases. It may also be concluded that the rate of development of the curve is important.

TABLE 5
FAMILY HISTORY OF DIABETES

| Type of Curve | Number | Negative | | Hereditary | | Familial | | Total Positive | |
|---------------------------|--------|----------|-----------|------------|-----------|----------|-----------|----------------|-----------|
| | | No. | Per cent. | No. | Per cent. | No. | Per cent. | No. | Per cent. |
| All curves | 2280 | 1825 | 80.0 | 326 | 14.4 | 129 | 5.6 | 455 | 20.0 |
| Negative | 359 | 322 | 89.7 | 32 | 8.9 | 5 | 1.4 | 37 | 10.3 |
| Positive | 1467 | 1161 | 79.2 | 212 | 14.4 | 94 | 6.4 | 306 | 20.8 |
| High peak+slow rise | 454 | 342 | 75.4 | 82 | 18.0 | 30 | 6.6 | 112 | 24.6 |
| High peak only | 340 | 260 | 76.5 | 64 | 18.8 | 16 | 4.7 | 80 | 23.5 |
| Slow rise only | 114 | 82 | 71.9 | 18 | 15.8 | 14 | 12.3 | 32 | 28.1 |

TABLE 6
FAMILY HISTORY OF DIABETES

| Type of Curve | Number | Negative | | Hereditary | | Familial | | Total Positive | |
|---------------------------|--------|----------|-----------|------------|-----------|----------|-----------|----------------|-----------|
| | | No. | Per cent. | No. | Per cent. | No. | Per cent. | No. | Per cent. |
| | | | | | | | | | |
| Insurance Group | | | | | | | | | |
| All curves | 377 | 281 | 74.5 | 70 | 18.6 | 26 | 6.9 | 96 | 25.5 |
| Negative | 45 | 35 | 77.8 | 9 | 20.0 | 1 | 2.2 | 10 | 22.2 |
| Positive | 173 | 133 | 76.9 | 29 | 16.8 | 11 | 6.3 | 40 | 23.1 |
| High peak+slow rise | 159 | 113 | 71.1 | 32 | 20.1 | 14 | 8.8 | 46 | 28.9 |
| High peak only | 133 | 97 | 72.9 | 28 | 21.1 | 8 | 6.0 | 36 | 27.1 |
| Slow rise only | 26 | 16 | 61.5 | 4 | 15.4 | 6 | 23.1 | 10 | 28.5 |
| Non-insurance Group | | | | | | | | | |
| All curves | 1903 | 1544 | 81.1 | 256 | 13.5 | 103 | 5.4 | 359 | 18.9 |
| Negative | 314 | 287 | 91.4 | 23 | 7.3 | 4 | 1.3 | 27 | 8.6 |
| Positive | 1294 | 1028 | 79.5 | 183 | 14.1 | 83 | 6.4 | 266 | 20.5 |
| High peak+slow rise | 295 | 229 | 77.7 | 50 | 16.9 | 16 | 5.4 | 66 | 22.3 |
| High peak only | 207 | 163 | 78.8 | 36 | 17.3 | 8 | 3.9 | 44 | 21.2 |
| Slow rise only | 88 | 66 | 75.0 | 14 | 15.9 | 8 | 9.1 | 22 | 25.0 |

In the next investigation an attempt was made to study the course of events in individuals in whom sugar was discovered during examination for life assurance and whose blood sugar time curves showed no abnormalities other than a high peak or a slow rate of development of the peak. As previously stated, some of these individuals were given diets to improve carbohydrate tolerance, while others received no treatment. Blood sugar time curves were then obtained at approximately 6 month intervals. Many cases were so observed, but 235 only are included in this report, because some did not follow treatment prescribed carefully, while others failed to return for further observation.

From a similar study in 1926 (Unpublished: Personal communication to Dr. E. P. Joslin) I concluded that the peak of the blood sugar time curve was a reliable index in the diagnosis of diabetes. At that time, however, the methods of treatment introduced a disturbing variable. Experiences since have shown that, when individuals who were normal or subnormal with respect to body weight are subjected to diets of low carbohydrate content, carbohydrate tolerance is impaired rather than improved in many cases. Since then, our diets have been more liberal with respect to carbohydrates. Treatment was changed from low carbohydrate-high fat to high carbohydrate-low fat diets. With this change, the data are more conclusive. They are summarized in Table 7.

It will be observed that of 235 individuals whose records are sufficiently complete to include in this study, the first curves and their incidence were as follows:

| GROUP | TYPE OF CURVE | NUMBER |
|-------|---|--------|
| 1 | Positive | 83 |
| 2 | Negative | 21 |
| 3 | Negative except for high peak | 79 |
| 4 | Negative except for slow rate of development of peak | 52 |

If we include the 131 doubtfully positive curves, because of the peaks, with the 21 which were definitely negative, it will be observed that, approximately, 64 per cent. of these individuals

TABLE 7
EFFECT OF DIET UPON BLOOD SUGAR TIME CURVES

| Group | Original Curve | Treatment | Number | Subsequent Curves | | | |
|-------|----------------|---------------|--------|-------------------|----------|-----------|-----------|
| | | | | Negative | Positive | High peak | Slow rise |
| 1 | Negative | (a) Given | 8 | 7 | 0 | 1 | 0 |
| | | (b) Not given | 13 | 2 | 7 | 3 | 1 |
| 2 | Positive | (a) Given | 61 | 3 | 18 | 32 | 8 |
| | | (b) Not given | 22 | 0 | 18 | 3 | 1 |
| 3 | High peak | (a) Given | 41 | 18 | 4 | 16 | 3 |
| | | (b) Not given | 38 | 4 | 17 | 8 | 9 |
| 4 | Slow rise | (a) Given | 20 | 9 | 5 | 4 | 2 |
| | | (b) Not given | 32 | 3 | 7 | 14 | 8 |

would have been regarded as normal, as far as their carbohydrate metabolism is concerned, in spite of the fact that there was a history of glycosuria in every case. The later curves are, therefore, of interest here, especially when they are related to treatment.

Cases could be cited from this group in which the individuals, when given no treatment, developed frank diabetes. Thus: Mr. J. P. D., Hosp. No. 2083/33 was examined in January, 1931, and the following blood sugar time curve was obtained:

| TIME | | BLOOD SUGAR (per cent.) |
|--------------------------------|-------|----------------------------|
| In the fasting state | | 0.108 |
| At the end of 30 minutes | | 0.200 |
| " | 60 " | 0.216 |
| " | 120 " | 0.120 |
| " | 150 " | 0.106 |

No attention was paid to the peak nor the rate of its development by the Company and he was accepted as a standard policyholder. In October, 1932, he complained of dimness of vision. A blood sugar examination was made and found to be 0.246 per cent. in the fasting state. He received no treatment. Six months later he became an active diabetic and was admitted to the hospital on April 10th, 1933, with sugar and acetone in the urine. He is now on a quantitative diet and the urine is kept free of sugar with difficulty with 20 units of insulin a day; there is usually hyperglycæmia in the fasting state. Other equally impressive cases could be cited, but I do not wish to make the statistical error of drawing conclusions from isolated cases. From the combined data, however, it will be observed that, in the curves in which the peak was the only abnormality, its presence was due to disturbance of carbohydrate tolerance.

I should like to deal firstly with the negative curves. It will be observed that of the 8 individuals (Group 1a) with negative curves who received treatment, the curves have remained negative in 7 cases; whereas, of the 13 individuals (Group 1b) who were not given treatment, the curves eventually became definitely positive in 7 cases; four are doubtful, and only 2 have remained

negative. It is my impression that the negative curves in these 21 individuals were largely due to slight change of diet (restriction of carbohydrates) prior to the test.

Of the 61 individuals (Group 2a) whose blood sugar time curves definitely indicated disturbance of carbohydrate metabolism and who received treatment, eighteen only have definitely positive curves at present; whereas, of the 22 (Group 2b) who received no treatment, the curves of nearly all—eighteen—are still positive; four are doubtful; *none are, as yet, negative*. It is to be noted, if the peak is an indication of disturbance of carbohydrate metabolism, forty individuals of the treated group still have some disturbance, though their conditions have improved. This fits in with the general experience in the treatment of frank diabetes.

What was the course of events in the doubtful cases, that is, with those curves in which the peak was the only abnormality? It will be noted that of 41 individuals (Group 3a) whose curves showed a high peak and who were given treatment, the curves are now definitely positive in 4 cases only; eighteen are negative and 19 are still doubtful; whereas, in the 38 cases (Group 3b) in which no treatment was given, 17 are now definitely positive; four only are negative and 17 are still doubtful.

The cases with the slow rate of development of the peak are not convincing. The percentages of definitely positive curves are, approximately, the same with, and without, treatment. It is, however, of interest to note that of 52 of these individuals, 12 eventually showed definitely positive curves.

Limited significance must, of course, be attached to these data, because of the smallness of the groups, but, combined with the experience of family history cited, I believe we have further evidence to support the view that the peak is important in the interpretation of the blood sugar time curve.

I have attempted to demonstrate that the blood sugar time curve, assuming that it has been properly obtained and properly interpreted, is one of the most valuable methods of detecting diabetes in its very early states. An additional advantage of this test is that it may detect diabetes in individuals in whom the renal threshold for glucose may be increased. The degrees of

hyperglycemia which may be met with in the absence of glycosuria are, at times, striking. In a recent study of this phenomenon (Bioch. J., vol. 26, p. 963, 1932), our records showed a blood sugar of 0.825 per cent. without glycosuria, and it was practically all due to glucose; very little—0.071 per cent. only—could be accounted for by non-fermentable reducing substances in the blood. As sudden or radical changes in diet are some of the conditions which may lead to a raised renal threshold for glucose, this phenomenon is of more than academic interest in life assurance work.

Valuable as the blood sugar time curve may be, it should not, however, in my opinion, be used to the exclusion of the simple urine examination for sugar, for, as I shall show, some of the practices in the past still have their uses, when properly obtained blood sugar examinations are not readily available. In this investigation, an attempt was made to determine whether there was a relationship between the type of glycosuria and the type of blood sugar time curve.

In 1924, Cook (Proc. 14 Annual Meeting of the Medical Section American Life Convention) quoted Professors Folin and Benedict as having made the observation that as much as 0.3 per cent. of sugar in the urine may be disregarded; that such applicants may be considered as standard, without further investigation. It is not clear as to whether this percentage referred to glucose or merely to the degree of reduction of alkaline-copper solution (Benedict or Folin) which corresponds to 0.3 per cent. glucose. One can readily agree with this observation if it applied to non-glucose reducing substances in the urine. Such a finding is common and appears to have no pathological significance. The findings of Doctors Exton and Rose of the Prudential Insurance Company of America are of interest here. Of 4,837 urines which gave slight but definite reductions with Fehling's and Benedict's solutions and which were subjected to the dinitrosalicylic acid and phenylhydrazine tests, 2,266—an incidence of 47 per cent.—showed no glucose, though the amounts of reducing substances ranged between 0.3 and 0.5 per cent. I cannot, however, agree with this observation attributed to Drs. Benedict and Folin, if it refers to glucose.

You may recall the warning of Dr. Joslin in 1921 that though a positive Benedict test does not prove diabetes, sugar in the urine should be regarded as due to diabetes, until proven otherwise. This audience may also recall Dr. Knight's report in 1921 which showed that in a group of individuals with glycosuria, even on one occasion only, the ratio of actual to expected deaths was high at practically all ages. This fits with Dr. Braithwaite's study in which a distinct increase in mortality was found with increase in the frequency of glycosuria. Dr. Braithwaite took a group of individuals where at least 4 samples were examined each three hours after a meal. In spite of the many variables which tend to minimize the value of such practice, it has its place, for I shall attempt to show, it may, with slight modification, be made to yield information not only approaching that of the blood sugar time curve, but is better than the blood test unless the latter is performed under proper conditions. With extremely few exceptions, glycosuria, in amounts detectable by the ordinary methods in clinical use, is still a *sine qua non* in the diagnosis of diabetes. Whether an extremely small amount of sugar detectable by special methods only is a normal constituent of urine, as Benedict contends, or whether it is due to absorption and excretion of foreign materials present in grain, vegetables and fruits, and decomposition products due to cooking, canning and baking of foods, as Folin suggests, is still largely controversial. Evidence is rapidly accumulating that glucose is a normal constituent of urine, but the amounts are extremely small^(1,2,3.). This, at present, is of academic interest only. We are here concerned with amounts detectable by the usual alkaline-copper reagents and I am quite in agreement with Dr. Joslin's observation that such individuals should be regarded as diabetics until proven otherwise. I have always contended that renal glycosuria is rarely met with when proper criteria are used for its diagnosis. A more recent reinvestigation in this Clinic of the literature and our own data (A. F. Fowler, *Ann. Int. Med.*, In Press) adds support to

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1. West, *Bioch. Jour.*, vol. 26, p. 1720, 1723, 1742; 1932.
 2. Hardin and Selby, *Bioch. Jour.*, vol. 26, p. 957; 1932.
 3. Hassan, *Bioch. Jour.*, vol. 22, p. 1332; 1928.

this view. It must be emphasized that absence of clinical signs and symptoms of diabetes does not exclude diabetes. Campbell⁽¹⁾ reports a condition designated as a 'diabetes innocens'. A group of cases were observed with no signs or symptoms of diabetes other than glycosuria; a long duration of glycosuria without development of symptoms in spite of no diatetic control; with normal fasting and post prandial blood sugars, but with blood sugar time curves approximately those of mild diabetics; increase of glycosuria without increase of food, but not proportionate; and high respiratory quotients. Such cases are occasionally seen in clinics for diabetes; but the condition is, in my opinion, not harmless. Infection of any kind may readily cause rapid loss of carbohydrate tolerance and, in the absence of treatment, lead to coma. I am quite in agreement with Dr. Joslin's observation that, though diabetes may be mild, there is no such thing as innocent diabetes.

To test the reliability of urinalysis alone, for the diagnosis of diabetes, 308 individuals were investigated, many of whom belong to the insurance group. Careful histories were obtained with regard to the type of the glycosuria. Our Routine History Form contains the following with respect to glycosuria:

Was sugar ever found in the urine? If so, complete the following details:

| Date | Hours | Before | After | Breakfast | Noon meal | Evening meal |
|-------|-------|--------|-------|-----------|-----------|--------------|
| | | | | | | |

The above data made possible the following classification of cases:

Group 1. Sugar found in a *random* sample; no definite statement was made as to the time of the day the urine was collected. No other examinations were made.

Group 2. Sugar found on one occasion only. Urine was collected shortly after a meal.

Group 3. Sugar found on two or more examinations and in each case shortly after a meal.

Group 4. Sugar found in the fasting state.

1. Campbell, W. R., J. A. M. A., vol. 82, p. 1990; 1924.

The above classification was based upon the usual course of the chronic progressive diabetic. In this type of diabetic, it will be recalled, the glycosuria is at first *transitory*; after its discovery it may disappear and not appear again for days or weeks. As the disease progresses, glycosuria is found fairly regularly, but after meals only—*post prandial glycosuria*. With further downward progress, the glycosuria becomes *persistent* and is then found in the fasting state.

To correlate the urine and blood tests, a composite blood sugar time curve was made of each group and to determine whether differences noted, if any, between the Means of the corresponding periods of the different curves were significant, calculations were made of the Ratios of the Differences between the Means to the Probable Errors of the Differences. The combined data are shown in Table 8.

There are a number of sources of error in the classification of these individuals. For example, the random samples may have been urines collected either in the fasting state or after meals; the post prandial samples may have been collected after carbohydrate-rich or carbohydrate-poor meals; and it does not necessarily follow that urines collected in the fasting state, before breakfast, actually represented the metabolism in the fasting state; some of these individuals may not have voided after the evening meal nor during the night. Therefore, some of the samples may have been urine partly of the fasting state and partly influenced by the meal the evening before—post prandial urine. However, it was for this reason that the Probable Error concept was made use of. A brief digression may, therefore, here be made with regard to Probable Error.

Let us take, as an example, the column in Table 8 in which are recorded the blood sugars at the end of 60 minutes. It will be observed that the average blood sugar in Curve 3 is 0.173 per cent. and in Curve 4 it is 0.181 per cent. The difference between the two blood sugars is, therefore, small; it is 8 milligrams only. This difference, it may be noted, could, in isolated cases, readily be due to the error inherent in the test, considering the height of the blood sugar. Are we, therefore, justified in

TABLE 8
Showing Relationship Between Type of Glycosuria and Type of Blood Sugar Time Curve

| Type of Glycosuria | Number of Cases | Fasting | | 30 minute | | 60 minute | | 120 minute | | 150 minute | |
|--------------------|-----------------|--------------------------|--------------------------------------|--------------------------|--------------------------------------|--------------------------|--------------------------------------|--------------------------|--------------------------------------|--------------------------|--------------------------------------|
| | | Blood Sugar Per cent. | *Ratio of Difference to its P. E. | Blood Sugar Per cent. | *Ratio of Difference to its P. E. | Blood Sugar Per cent. | *Ratio of Difference to its P. E. | Blood Sugar Per cent. | *Ratio of Difference to its P. E. | Blood Sugar Per cent. | *Ratio of Difference to its P. E. |
| 1 | 161 | 0.103 | 3.5 | 0.192 | 2.1 | 0.153 | 3.3 | 0.131 | 6.1 | 0.111 | 4.2 |
| 2 | 82 | 0.128 | 1.5 | 0.220 | 4.2 | 0.166 | 5.2 | 0.143 | 2.6 | 0.126 | 2.1 |
| 3 | 49 | 0.131 | 3.1 | 0.232 | 3.9 | 0.173 | 4.1 | 0.153 | 6.4 | 0.139 | 3.53 |
| 4 | 16 | 0.120 | | 0.246 | | 0.181 | | 0.166 | | 0.137 | |

*Ratio of Difference to its Probable Error = $\frac{\text{Difference between Means}}{\text{Probable Error of Difference}}$

concluding that the average blood sugar is higher in Curve 4 than in Curve 3? Are we justified in concluding that there is a relationship between the type of glycosuria and the blood sugar 60 minutes after ingestion of glucose, or is the 8 milligram difference due to the operation of the Laws of Chance? Whether this difference of blood sugar is, or is not, significant may be determined by its Probable Error. It will be noted that the Ratio of the difference between these two blood sugars to the Probable Error of the difference is over 4 (4.1 to be exact). According to this Ratio, it can be shown that the odds against this difference of 8 milligrams being due to chance only are about 175 to 1. As another example, let us examine the 120 minute column. It will be noted that the difference between the average blood sugar of Curves 3 and 4 is 13 milligrams and that the Ratio is more than 6 (6.4 to be exact). From this, it can be shown that the odds against the occurrence of such a difference of blood sugar being due to chance are over 20,000 to 1. In other words, the higher the Ratio, the more is the element of chance eliminated. The results of this investigation may, therefore, be summarized briefly. The combined data of Table 8 led to the following generalization:

If urine is carefully examined in the fasting state and in relationship to ordinary meals; and if the type of glycosuria is thus carefully established (that is, whether it is transient, post prandial or constant), the information obtained is practically equal to that of the blood sugar time curve. At times, it may yield more valuable information since, compared with blood sugar examination and interpretation, urinalysis is a simple clinical procedure. These data also support the practice of some companies which do substandard insurance; that is, they support the rule of declination of individuals with persistent or intermittent glycosuria. Incidentally, the Ratios in the 30 minute column of this Table further emphasize the importance of the peak of the curve. It will be observed that for Curves 2 and 3, the Ratio is 4.2 and for Curves 3 and 4 it is 3.9. The odds against these differences of blood sugar being due to chance are over 100 to 1.

In 1930, I had the occasion to discuss pitfalls in the clinical application and interpretation of basal metabolic rates. In my

closing remarks, I referred to one of John Brown's delightful essays. It is equally applicable here. You may recall the brisk dilettante student saying to the great painter:

"'Pray, Mr. Opie, may I ask what you mix your colours with?' 'With brains, Sir,' was the gruff reply—and the right one. It did not give much of what we call information; it did not expound the principles and rules of the art; but, if the enquirer had the commodity referred to, it would awaken him, it would set him a-going, a-thinking, and a-painting to good purpose. If he had not the wherewithal the less he had to do with colours and their mixtures, the better." This applies equally well to the use of blood sugar time curves.

DR. MCCRUDDEN—The mortality of glycosuria subjects selected on the most careful basis possible up to ten years ago was excessive. The proportion of deaths from diabetes among those selected was many times that among standard risks. The mortality of glycosuria subjects showing not more than 0.12 per cent. blood sugar two hours after ingestion of 100 grams glucose and insured as standard risks by the New England Mutual during the past ten years has been no greater than that of the general business of the Company during the same period. There has been no death in the group from diabetes and no increase in the proportion of deaths from disease of the circulatory system or from acute infections that might suggest a diabetic background.

Dr. Rabinowitch's contributions to our understanding of this important blood-sugar tolerance test are outstanding. His ingenious simultaneous determinations of the respiratory quotient, the arterial blood sugar, and the venous blood sugar have thrown much light on the happenings after glucose ingestion. His comments regarding the significance of the various features of the test and the factors which influence those features are well grounded. His conclusions as to the application of the test in the diagnosis of diabetes are sound. His stress on the necessity of care in the administration of the drink, in the operation of

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taking the blood sample, and in the carrying out of the analysis deserves close attention and hearty applause.

In applying clinical findings to underwriting we should remember that clinical medicine is not underwriting. The clinical problem presented by a medical impairment is not the same as the underwriting problem. The purpose of clinical examination is not that of insurance examination. The objective of the patient is not that of the insurance applicant. The technical requirements of an insurance examination will not be the same as those of a clinical examination.

Clinical medicine deals with the diagnosis and treatment of disease; underwriting deals with the appraisal of the insurance value of risks. The clinician asks: "Which glycosuria subjects have diabetes?"; the underwriter asks: "Which of them are standard risks?" These are two different problems.

Clinical examination aims at *individualization* of the subjects examined; insurance examination at *grouping* them. Individualization implies the numerous, complicated tests of an exhaustive examination. Grouping implies a minimum of simple tests. In examining glycosuria subjects the repetition of tests required for determination of the influence of different amounts of ingested glucose on the form of the curve, the repetition of venesections required to determine rate of rise and rate of decline in the curve, the control over the subject's time and activities required for stipulation of such details as time of day, muscular activity, previous diet, desirable as they may be for individualization, are not only impractical but unnecessary for grouping.

The clinician's subjects are patients; the underwriter's subjects are insurance applicants. The patient wants a diagnosis. The insurance applicant wants a policy. The insurance applicant will not play the same passive part in the examination as the patient. In the hope of replacing blood analysis as a basis of grouping with the simpler urine analysis, Rabinowitch attempted to correlate type of glycosuria with type of blood-sugar tolerance test. He showed that the differences between the average or composite form of blood-sugar tolerance curves associated with certain types of glycosuria are so great in com-

parison with the probable errors of those average curves that the differences must be significant. He concluded that urine analysis can be nearly, if not quite, as dependable as blood analysis for evaluating the significance of glycosuria. His method of investigation is mathematically sound, but his conclusions will be applicable in practice only if future samples of the groups studied are similar in composition to his test samples. If the membership of future samples is determined as that of his test samples was, by random selection, those samples will be similar in composition to his test samples and his conclusions will apply. If the membership of future samples is not determined wholly by random selection, but is influenced by some other force, future samples will not be similar in composition to the test samples and his conclusions will not apply. There is no reason why the membership of future *clinical* samples should not be determined, just as that of the test samples was, by random selection. Such samples should be similar in composition to his test samples and his conclusions should apply in clinical practice. Experience with groups which are not homogeneous has shown that the membership of *insurance* samples is not wholly determined by random selection; other forces in the form of special selfish interests—we call these the forces of antiselection—do play a part, often a large part in determining the membership of insurance samples. Such samples will not be similar in composition to clinical samples. The conclusions warranted as applicable in clinical practice will not be applicable in insurance practice.

In view of the difference in the problem, in the purpose of examination, and in the objective of the subject examined, the technical requirements of an insurance examination will not be the same as those of a clinical examination. The facts to be gathered in the examination, the requirements as to time consumed, expense, and convenience in the gathering of the facts, the stipulations as to conditions surrounding the gathering of the facts, and the interpretation of the facts will all be different in the two cases.

Discussion—The Diagnosis of Diabetes 47

The medical aspect moreover is only one aspect of an underwriting problem; the medical hazard often is not the only hazard encountered. The insurance value of a risk has social, moral, and financial components as well as medical components. In the glycosuria group the non-medical aspect is especially prominent.

The mortality by policy count in the New England Mutual glycosuria group was only 84 per cent. of that by amount. Policies for \$50,000 or more, making up only $3\frac{1}{2}$ per cent. of the total number of policies issued to the group, accounted for 14 per cent. of the claims by policy count and 60 per cent. by amount. Individual lives insured for \$100,000, representing only $12\frac{1}{2}$ per cent. of the total claims by lives, accounted for 60 per cent. of the claims by amount. The adverse influence of large policies, known to be great in any group, would appear to be exaggerated in the glycosuria group.

Suicide accounted for 20 per cent. of the losses in the group by lives, and 36 per cent. by amount. Violent deaths of some kind accounted for 38 per cent. of the losses by lives, and 59 per cent. by amount. The proportion of suicide and other violent deaths is far greater than in our general business. The combination of glycosuria and large amount of insurance seems to present a particularly great suicide and violent death hazard.

Seventy-five per cent. of the claims in this group were on executives, bankers, brokers, and professional men. The average age at entry of the losses was 48.

The blood-sugar tolerance test used as we use it appears to have overcome the medical hazard in the glycosuria group. But the proportion of losses from suicide and other violent deaths among middle-aged brain workers carrying large policies was excessive. There still remains a definite non-medical, environmental hazard. Glycosuria is a good example of the difference between the clinical problem and the underwriting problem presented by a medical impairment.

DR. GORDON—What I have to say after Dr. Rabinowitch's paper will come as an anticlimax. I have had the opportunity of watching this work from the day Dr. Rabinowitch undertook

it in the Montreal General Hospital and I may say, as an interested onlooker, I have seen the inception, the development and the completion of what we have heard today and I feel very proud of it.

There are some things I might mention and this is one: in reference to the point Dr. Rabinowitch has brought out in regard particularly to the peak and the time of its appearance, I first viewed this with some skepticism but as time has gone on, I feel from our clinical experience that these observations have been more than justified.

It would be quite useless for me to attempt to make any criticism of these observations. I can only comment on two or three. One point that I think everyone in this room should make considerable of is the necessity, of doing blood sugar time curves at all, to paraphrase someone else's statement and say "If 'twere done, 'twere well 'twere done well." If a time curve is being done, one should be assured that the technician has not only brains, but a consciousness that the procedure should in all cases be standard. It seems to me that it would be very wise for us to adopt soon some standard method by which these sugar time curves are carried out.

Again, the observation I think one might comment upon is that well taken and carefully examined periodic specimens of urine taken in the fasting state and after a meal do seem to offer, if not exactly the same, at least a very similar type of security to the blood sugar time curves as taken by the present method. When standardization is more complete, when one is more certain of the place where the specimen is taken and examined, I think, possibly, we may look for better generalization in sugar time curves than we find in the examination of urine now. At present however the careful examination of urine has almost, if not altogether the same value as has the taking of sugar time curves.

I have been delighted with Dr. Rabinowitch's paper and I have followed it with a very great interest. Thank you, Mr. President, for asking me to say a few words.

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DR. EXTON—I think that we should all of us feel very grateful to Dr. Rabinowitch for giving us in such a vivid way the benefit of his very rich clinical experience. Naturally, I read his paper with a great deal of interest and have tried, as I always do when reading papers that interest me, to find matters about which I might disagree. It is, therefore, very pleasant to confess that I did not find anything in Dr. Rabinowitch's paper with which one could seriously disagree. Perhaps just a quibble would be in order concerning his statement that the term "sugar tolerance test" is an erroneous designation of what Dr. Rabinowitch prefers to call a "blood sugar time curve". The difference between the two reminds me of a headline I read in a New York paper the other day about a man who was killed while trying to commit suicide.

Whatever may be the difference between macro and micro methods, there is no room for any disagreement about the matter of our being obliged to rely on micro methods in doing life insurance work simply because the macro methods are impracticable. It would be a wonderful thing if we could all of us send all of our applicants to Dr. Rabinowitch's or some other laboratory that was just as reliable as his. Unfortunately, that is impossible because our cases occur all over the country.

This circumstance has given me an opportunity to compare many curves of the same people made by the same method, both micro and macro, in different laboratories. The comparisons show many discrepancies as great or even greater than the example of Dr. Birchard's which Dr. Rabinowitch gives, even when the different laboratories employed the same micro or macro method. There are so many details which enter into the accuracy of results that it seems to me to be merely a matter of one laboratory being reliable and another unreliable. This looks to me to be probably the case in the single illustration which Dr. Rabinowitch gives and which I, therefore, think is not a fair and complete demonstration of any advantage of the macro over the micro blood sugar tests. It is a good chance, however, to emphasize what Dr. Rabinowitch has so truly said: "Whatever method is used must be well done".

It is now possible for insurance offices to make all their blood sugar determinations in the home office laboratory without depending upon examiners for more than merely pricking the finger and letting a few drops of blood fall into a glass or hycoloid vial. In this way all of the measurements, calculations and other details that enter into the accuracy of blood sugar determinations are under the control of a home office chemist who uses the same equipment and methods for all specimens. In our experience this is the only practicable way to avoid the inaccuracies and inconsistencies that seem to be inseparable from the reports received from different laboratories.

In concluding, I desire to thank Dr. Rabinowitch again for his very valuable and interesting contribution to our meeting and our knowledge of diabetes.

DR. RABINOWITCH—I quite agree with Dr. McCrudden with respect to the possible error in the assumption that collection of urine is random and in the assumption that a sample alleged to be post-prandial is really urine collected after meals. Fortunately, the relationship between physician and patient is not exactly that of insurance company and applicant. I appreciate your difficulties and thank my stars that I am not a Medical Director.

With regard to Dr. McCrudden's observations on the relationship between mortality and blood sugar two hours after administration of glucose, it would be of interest to know what method Dr. McCrudden uses for the estimation of blood sugar. I believe that the majority of laboratories are now using the more recent methods which yield lower values than the older procedures. A blood sugar of 0.12 per cent. is not a normal blood sugar according to the new tests and when found two hours after administration of glucose has a different meaning than the same blood sugar with the older methods. In view of the lower values with the newer methods, I believe it a reasonable assumption, when the blood sugar obtained with the newer methods is 0.12 per cent. two hours after administration of glucose, that the peak of the curve was quite high. If, therefore,

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Dr. McCrudden uses the newer methods his data would fit with my own.

With respect to Dr. Gordon, I should like to add to your remarks, Mr. Chairman, and to those of Dr. Gordon. Dr. Gordon has not only been watching the progress of the Metabolism Laboratories of The Montreal General Hospital, but was very largely responsible for their institution and to Dr. Gordon must be credited much of the work which is being done. He was, and still is, my teacher.

I was expecting some discussion by Professor Folin with regard to my remarks on micro methods for estimation of blood sugars. Professor Folin apparently agrees with what I had to say about the possible sources of error. As I stated, micro methods can be very exact. As with all other laboratory procedures, however, results depend upon the worker. I can readily see that micro methods are more practical than the macro procedures in insurance work. The number of factors, however, which govern accuracy must be considered. In view of the sources of error, it is my impression, Mr. Chairman, that if these micro-methods are to be used by life assurance companies, results of tests should be accepted from well tested laboratories only or, probably still better, the work should be done by companies in their own offices and by technicians specially trained in these procedures.

DR. SCADDING—The second paper will be presented by Dr. Frederick G. Brathwaite, who is the sweetest and staunchest of friends. His wonderful shop is now located in the Metropolitan area of New York, where he has had abundant opportunity of "riding his hobby"—the study of glycosurics. He'll show you how skillfully he does it.

GLYCOSURIA: A SELECTIVE FORMULA AND ILLUSTRATIVE MORTALITY STUDY

BY FREDERICK G. BRATHWAITE, M. D.

Equitable Life Assurance Society, New York City

When it became necessary for us to revise our ideas in prognosis of cases of glycosuria due to the development in the study of blood chemistry, we cast about for some method in which we could give certain mortality "weight" derived from the information obtained by sugar tolerance tests.

We were not prepared to abandon the criteria already in use based on the study of a voluminous amount of material in connection with urinary findings.

In 1924 Allen ⁽¹⁾ said that the significance of any given symptoms from the statistical standpoint could be established only by insurance companies. He further stated that it might be assumed that whether or not diabetics were to be granted insurance, the company was at least desirous of knowing if diabetes were present at the time of the examination. This was necessary in order to make an intelligent decision, it being understood that there is a limit to the complexity of the examination. For example, he said the practical decision is generally based on urinalyses with or without the aid of carbohydrate test meals and blood analyses are generally considered impracticable for routine use, admitting that it was without dispute that many diagnoses were missed on this basis.

We were convinced, however, that blood analyses were *not* generally impracticable and from 1925 made them a part of our routine selective process in glycosuria, except for small amounts of insurance.

The question then arose as to what values to allot to the incidence and degree of glycosuria as affected by the results of

(1) Proceedings of the 14th Annual Meeting of the American Life Convention, "Glycosuria and Diabetes", Allen, Frederick.

sugar tolerance tests, favorable or otherwise, assuming that both should be taken into consideration. Here it is appropriate to point out that, as Dr. Rabinowitch explains, these tests may be more properly designated blood sugar time curves.

At the outset a difficulty arose from the apparent conflict between the concept of the condition held by the clinician and that held by the Life Insurance Company based on mortality studies. The former is usually confronted with the problem of determining whether the glycosuria is of serious significance or not. The insurance company is concerned with glycosuria either accidentally discovered in apparent health or known to have existed more or less constantly over a period of years without giving rise to symptoms.

Various writers have drawn attention to the fact that the public owes a debt to the insurance companies in discovering glycosuria in an individual in whom it may prove more or less serious at a later date. No less an authority than Joslin ⁽²⁾ has said "A medical examiner and the doctor interested in diabetes have much in common", adding that the work of each is largely of a statistical nature and success or failure (in the solution of the problem) is almost entirely based on the courtesy employed in the gathering of statistics and their interpretation.

Herein lay another difficulty inasmuch as the interest of the insurance expert lies in the final outcome ("exit") of cases observed, namely death; while the outcome ("exit") in clinical statistics did not and could not mean death invariably, because the clinician can record in his statistics either the date of death or the exposure from a life insurance standpoint with comparative infrequency.

Our point of view was, however, based on the hypothesis that it was essential to carry the statistical method to the limit, bearing in mind the facts above outlined which are impressive when a large number of cases are studied on the basis of exposure, from the date of entrance in a given group until death. We assumed, therefore, at the beginning, somewhat empirically, that the basis for a prognostic formula should take into consideration

(2) Thirty-second Meeting of the Association of Life Insurance Medical Directors, 1921. Joslin, Elliott.

a valuation ("weighting") for mortality which should include both urinary and blood sugar findings. We believed that a penalty could be imposed which would represent a combined expression of the importance attached to each factor, without reliance exclusively upon either one or the other.

Starting then, with glycosuria as the primary evidence of abnormality, we constructed a table which had for its basis the incidence of glycosuria and the amount of glucose determined by Home Office quantitative analysis of urinary specimens, taking care to exclude other substances giving a reducing reaction by always employing the phenylhydrazine and other tests.

We designated the incidence A, B, C and D according to whether it was accidental, occasional, intermittent or constant and cases were placed in one of these four classes and given a value in figures which showed progressively increasing importance in comparison with the total number of specimens examined or shown in the insurance record within three years. Setting these letters in a vertical column we ran a horizontal line opposite each letter also containing four letters E, F, G and H, dependent upon whether the amount of sugar in the urine on the average was less than .3%; .3% to .49%; .5% to .99% and 1.0% and over.

In our computation of the incidence of glycosuria we adopted a fractional method of expressing the same; that is, $\frac{3}{5}$ meaning three positive findings out of five specimens examined and/or included in the insurance record within three years. In computing the amount of sugar found we adopted for reasons that will be seen, two averages, one which we call the "whole" average indicating the average amount of sugar found in all specimens including negative ones, which were being considered in the computation, and a second average called the "plus" average which is the average amount of urinary sugar only in those specimens which were positive for sugar, omitting from the computation those specimens which were negative for sugar.

This was an arbitrary method of averaging the sugar but served a purpose by which when blood sugar observations were favorable we used the more favorable or "whole" average for

urinary sugar theoretically to offset the penalty which would be called for by the not infrequently high urinary sugar findings, supposedly due to the ingestion of glucose.

Taking, therefore, the vertical column as expressive of incidence and the columns intersecting the horizontal lines opposite the four letters as expressive of amount, we erected the following table, inserting specific plus ratings at the points of intersection of the column and the line:

TABLE 1

| | E | F | G | H |
|-----------------------|-------------------|------------------|------------------|-------------------|
| | Less than 0.3% | 0.3% to 0.49% | 0.5% to 0.99% | 1.00% and over |
| A Accidental | +5 | +10 | +20 | +40 |
| B Occasional | +10 | +20 | +40 | +80 |
| C Intermittent | +25 | +50 | +100 | +200 |
| D Constant | +40 | +75 | +150 | +300 |

Thus the letters CF, for example, mean that in a given case an applicant who showed intermittently a urinary sugar content of from .3% to .49% was to be charged an extra premium sufficient to cover a mortality rate 50% in excess of that on standard lives.

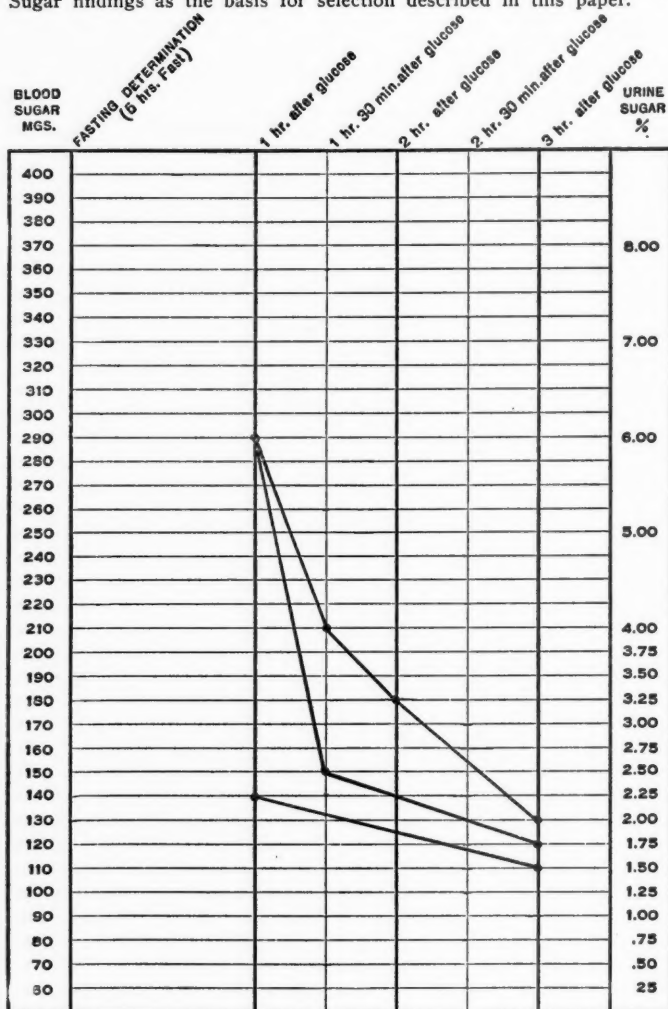
The numerical penalty was, we thought, severe, judging from our experienced mortality and was so intended in order to give the Society the benefit of any doubt and in order to rate cases somewhat heavily when urine alone was considered, in order to encourage blood sugar observations and to protect us against those cases actually or potentially diabetic, notwithstanding findings of a low urinary sugar content.

An arbitrary rule was then adopted that cases upwards of \$10,000 total at risk necessitated blood sugar observations. Hence it was necessary to bring the second element of the double criterion into play and we arranged a formula in which the first part of an equation consisted of two letters which indicated the incidence and amount of urinary sugar and the second part of the equation represented blood sugar values and was determined by the character of the blood sugar curve over a three hour period.

For the second part of the equation we allowed the blood sugar values to fall into four groups and called them plus,

TABLE 2

Outline of Blood Sugar areas used in conjunction with Urinary Sugar findings as the basis for selection described in this paper.



indeterminate, minus and double minus meaning that a sugar tolerance curve was either clearly diabetic, potentially or questionably diabetic, not diabetic and a curve indicating the active mobilization of insulin.

We then designed a chart on which the blood sugar findings in any specific case could be indicated by a curve. This chart consisted of three curves starting at a vertical line representing the beginning of the test and passing successively through points representing the end of the first, second and third hours.

The points through which these three curves passed represented in each curve successively higher ordinates as compared with a base line. Taken in conjunction with the base line, they, therefore, established four areas on the chart and the curve to be plotted in any specific case would fall entirely or preponderantly in one of these four areas.

These curves were *not intended to represent the clinical conception* of the four groups of sugar tolerance (above mentioned) inasmuch as this part of our computation represented only half the picture, the other half being represented by urinary sugar values which bore a certain relationship in our formula to the blood sugar values. Summing up, therefore, the theory on which this formula was constructed allowed the first element of an equation to represent incidence and the quantity of urinary sugar and the second element to represent the value of a sugar tolerance curve. The resultant obtained by equating these two elements, therefore, was expressed by a combination of two letters referred to in the table, the corresponding numerical values of which indicated our final evaluation of the penalty to be imposed, if any, from the standpoint of glycosuria.

The urinary sugar penalty having been allotted a value, that penalty was imposed if no blood sugar test had been done. If, however, a blood sugar test had been carried out, we then allowed the blood sugar curve to affect that value as follows:

If a blood sugar fell in the area above the highest curve ($a + \text{B.S.}$) it was invariably declined. If it fell in the next lower area (an indeterminate or $\pm \text{B.S.}$) the already determined urinary value was *unaffected* and the case was classed as potential

diabetes and rated according to the penalty provided for urinary findings alone. If it fell in the next lower area (a favorable or — B.S.) it acted as a *credit* to offset (somewhat) the penalty for predetermined urinary value. If it fell in the lowest area (a very favorable or — 2 B.S.) it acted as a more *marked credit* as being indicative of active insulin mobilization.

It was provided that if the blood sugar curve fell in more than one of the prescribed areas its preponderant significance be considered according to the area in which the curve fell most largely during the second hour. When we received a blood sugar and it was classified as — B.S. the urinary values were calculated on the "whole" average, instead of the "plus" average. If, however, we received a blood sugar which was — 2 B.S. therefore warranting further leniency, the urinary values were calculated on the basis of one-half the whole average instead of the plus average.

The above two methods of extending leniency in the ratings for the two varieties of favorable blood sugars were used until the current year at which time there were introduced adjustments, in tabular form, as shown in Table 4.

This was more convenient in office routine and in general approximated the leniency exercised under the method above described.

The effect of the above arrangement in which a separate "weighting" for urine and blood (double criterion) is provided for may be seen by the following tabulation:

Example DG

TABLE 3

| Urine | Blood | | Result |
|-------|--------|---------------|---------------|
| D G + | B. S. | (Blood Sugar) | = Declination |
| D G ± | B. S. | " " | = DG |
| D G — | B. S. | " " | = DF |
| D G — | 2B. S. | " " | = DE |

In other words we treat a plus blood sugar (+B.S.) as frank diabetes (declined), an indeterminate blood sugar (±B.S.) as potential diabetes, a minus blood sugar (—B.S.) as non diabetic and a double minus (—2 B.S.) as entirely normal which means, by reference to Table 1 that in a given case where the penalty

for urinary sugar was reckoned to be +150 this figure was affected by a minus or favorable blood sugar as described above, with the result that the altered value allotted was one-half that for the urinary value alone, viz., +75. For a double minus, or very favorable blood sugar, the penalty allotted was one-quarter that for the urinary value alone, viz., +40.

This method of selection was, therefore, as follows:

1. Ascertain from the urinary standpoint the incidence and quantity of glycosuria for the first part of the equation as described. If no blood sugar test has been obtained apply the proper rating penalty at this point.
2. When the blood sugar test has been obtained we then set up an equation as follows, and as shown in Table 3.
 - A. If the sugar tolerance was found to be frankly diabetic (+ B.S.) decline.
 - B. If the sugar tolerance was found to be indeterminate (\pm B.S.), use the same values as for glycosuria as though a blood sugar had not been obtained.
 - C. If the sugar tolerance was found to be favorable, ($-$ B.S.), use the values in the column $-$ B.S.
 - D. If the sugar tolerance was found to be very favorable (-2 B.S.), use the values in the column -2 B.S.

The combined table of values thus arrived at was as follows:

TABLE 4

| | E | | | F | | |
|----|----------------|----------|-----------|---------------|----------|-----------|
| | less than 0.3% | $-$ B.S. | -2 B.S. | 0.3% to 0.49% | $-$ B.S. | -2 B.S. |
| A. | +5 | +0 | +0 | +10 | +5 | +0 |
| B. | +10 | +5 | +0 | +20 | +10 | +5 |
| C. | +25 | +15 | +5 | +50 | +25 | +15 |
| D. | +40 | +20 | +10 | +75 | +40 | +20 |

| | G | | | H | | |
|----|---------------|----------|-----------|----------------|----------|-----------|
| | 0.5% to 0.99% | $-$ B.S. | -2 B.S. | 1.00% and over | $-$ B.S. | -2 B.S. |
| A. | +20 | +10 | +5 | +40 | +20 | +10 |
| B. | +40 | +20 | +10 | +80 | +40 | +20 |
| C. | +100 | +50 | +25 | +200 | +100 | +50 |
| D. | +150 | +75 | +40 | +300 | +150 | +75 |

Composite Sugar Tolerance Curves Illustrative of the Four Divisions of Sugar Tolerance Described Above:
Run Between October 1931 and December 1932 (15 months only) and Not Included in the Mortality Study.

TABLE 5

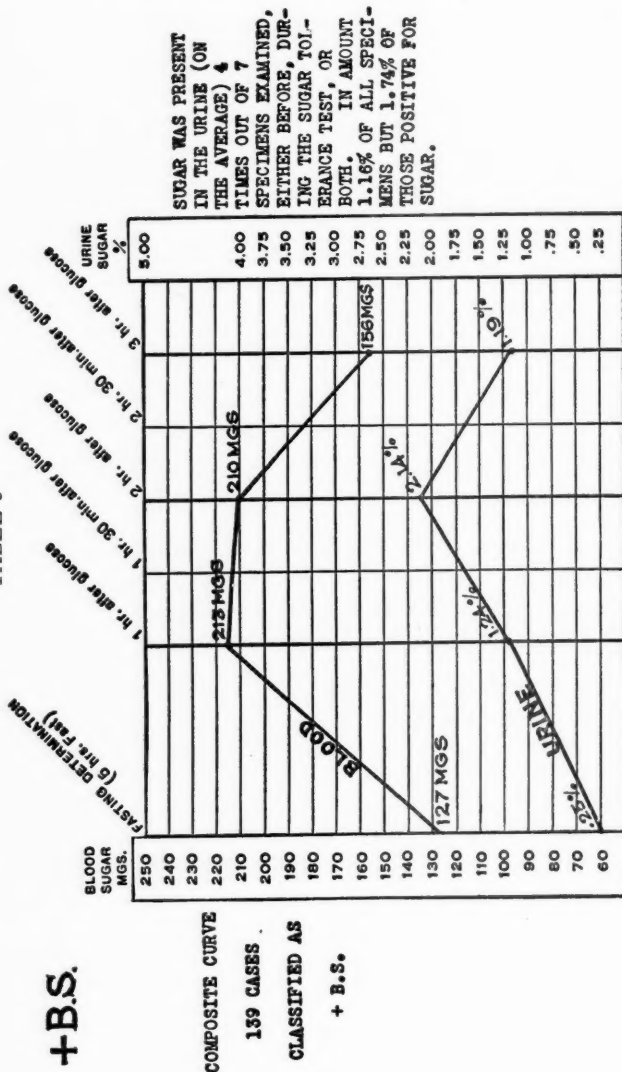
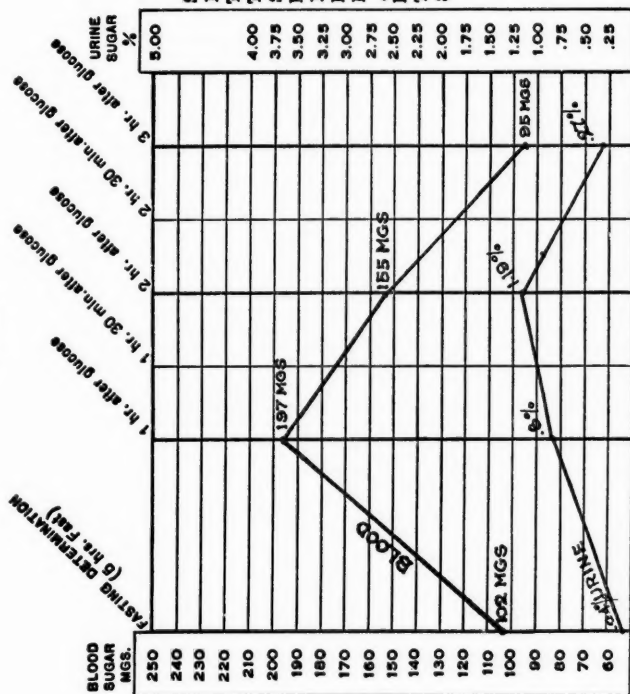
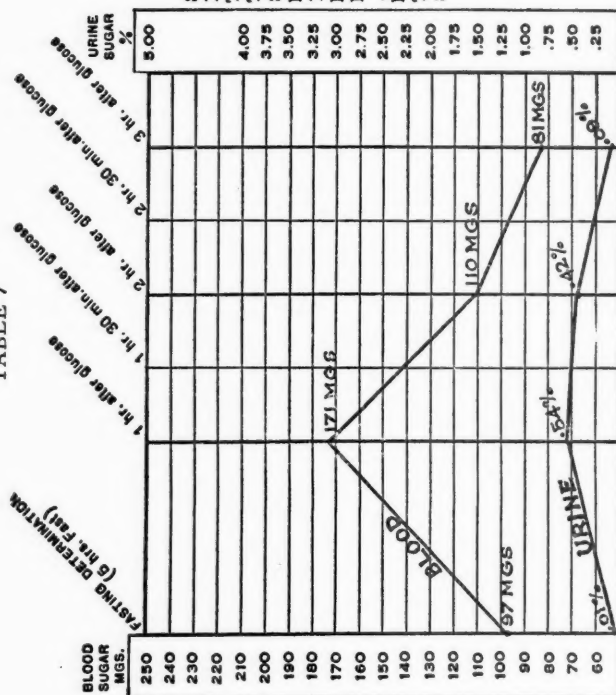


TABLE 6



SUGAR WAS PRESENT
IN THE URINE (ON
THE AVERAGE) 3
TIMES OUT OF 7
SPECIMENS EXAMINED,
EITHER BEFORE, DUR-
ING THE SUGAR TOL-
ERANCE TEST, OR
BOTH. IN AMOUNT
.55% OF ALL SPECI-
MENS BUT 1.01% OF
THOSE POSITIVE FOR
SUGAR.

TABLE 7



SUGAR WAS PRESENT
IN THE URINE (ON
THE AVERAGE) 2
TIMES OUT OF 7
SPECIMENS EXAMINED,
EITHER BEFORE, DURING
THE SUGAR TOL-
ERANCE TEST, OR
BOTH. IN AMOUNT
.23% OF ALL SPECI-
MENS BUT .73% OF
THOSE POSITIVE FOR
SUGAR.

- B.S.

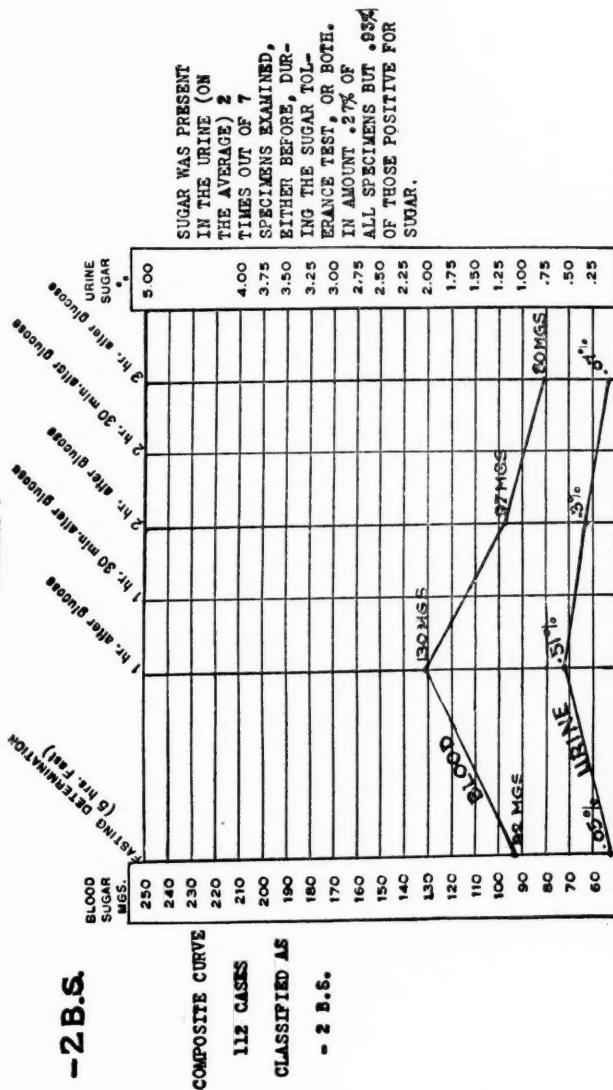
COMPOSITE CURVE

201 CASES

CLASSIFIED AS

- B.S.

TABLE 8



The technique of our sugar tolerance test is as follows:

- A. Fast of five hours.
- B. Take specimens of capillary blood and urine.
- C. Give 75 grams of glucose.
- D. At intervals of one, two and three hours following administration of glucose, secure specimens of both capillary blood and urine.

(NOTE: Subject is under observation during entire period of test.)

Urine and blood are examined at the Home Office Laboratory by the Folin Micro method and as far as possible by the same technician. Tests departing in any particular from the above are considered as being incompatible with a homogeneous collection of data. Any suspected glycolysis requires repetition of the test. All urine is examined for acetone and diacetic acid, as well as microscopically and glucose reactions are confirmed by phenylhydrazine and other tests.

CASES STUDIED FROM OCTOBER, 1925, TO DECEMBER 31, 1931

In the following mortality experience based on data collected and rated as above described, full sugar tolerance tests were run on 3,227 cases. The series includes cases issued from 1925 to 1931 with exposures carried to the examination anniversary in 1932.

The expected deaths were figured from a basic mortality table representing the standard experience on all cases from 1920 to 1926 observed to the 1927 anniversaries of companies contributing to the Joint Occupation Study 1928. This table probably represents more closely than any other available one the mortality on the standard lives of the Society during the period covered by the investigation alluded to.

VOLUME OF MATERIAL IN STUDY

The following compilation represents this volume

| | |
|--|-------------|
| I. Entrants in this study deemed to be standard lives..... | 2,346 |
| II. Entrants in this study deemed to be substandard lives..... | 2,263 |
| | <hr/> 4,609 |

A Study of Glycosuria

65

STANDARD EXPERIENCE BY LIVES

2,346 Entrants

| Issue Ages | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|------------|----------------|-----------------|---------------|-----------|
| to 39 | 4174 | 10.80 | 6 | 56% ± 20 |
| 40-49 | 2633 | 13.99 | 23 | 164% ± 23 |
| 50 & over | 1319 | 17.34 | 20 | 115% ± 17 |
| ALL | 8126 | 42.13 | 49 | 116% ± 11 |

| Policy Years | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|--------------|----------------|-----------------|---------------|-----------|
| 1 | 2346 | 8.51 | 15 | 176% ± 30 |
| 2 | 1852 | 9.09 | 8 | 88% ± 22 |
| 3-4 | 2692 | 15.95 | 16 | 100% ± 17 |
| 5-7 | 1236 | 8.58 | 10 | 117% ± 25 |
| ALL | 8126 | 42.13 | 49 | 116% ± 11 |

SUBSTANDARD EXPERIENCE BY LIVES

2,263 Entrants

| Issue Ages | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|------------|----------------|-----------------|---------------|-----------|
| to 39 | 2982 | 7.56 | 11 | 146% ± 29 |
| 40-49 | 2467 | 13.09 | 20 | 153% ± 23 |
| 50 & over | 1543 | 19.85 | 34 | 171% ± 20 |
| ALL | 6992 | 40.50 | 65 | 160% ± 13 |

| Policy Years | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|--------------|----------------|-----------------|---------------|-----------|
| 1 | 2263 | 8.99 | 13 | 145% ± 27 |
| 2 | 1604 | 8.77 | 15 | 171% ± 29 |
| 3-4 | 2134 | 14.25 | 19 | 133% ± 20 |
| 5-7 | 991 | 8.49 | 18 | 212% ± 33 |
| ALL | 6992 | 40.50 | 65 | 160% ± 13 |

| Mortality Group | Entrants | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|-----------------|----------|----------------|-----------------|---------------|-----------|
| 135-145% | 704 | 2190 | 12.67 | 18 | 142% ± 22 |
| 150-195% | 955 | 2964 | 17.11 | 23 | 134% ± 19 |
| 200-300% | 604 | 1838 | 10.72 | 24 | 224% ± 30 |
| ALL | 2263 | 6992 | 40.50 | 65 | 160% ± 13 |

The above study of mortality is composed of all cases which have shown sugar on examination or a history in the insurance record within three years, or both, but some cases giving evidence of other slight impairments, relative or not, have been included.

In considering the usefulness of the above outlined formula in the light of mortality experience obtained during its continuance, approximately seven years, we have been able to arrive at certain conclusions from the study of the material obtained during this period.

- A. It was obvious that wide difference of opinion existed in the consideration of glycosuric applicants when sugar tolerance tests entered into the record. For, in cases with insurance records of sugar tolerance, it was found on inquiry that many life companies were guided to a large extent by clinical opinion outside their own laboratory investigations. It so happened that there was at times a conflict with respect to the conception of the object sought. We should be clear as to our objectives. The clinician is primarily concerned with an opinion as to the existence of diabetes and then with treatment and prognosis. The life company is, or should be, interested in a more exacting study concerning the question as to whether or not a glycosuric is insurable. This involves a differentiation as between the existence of frank diabetes, or something else, that is, the prediabetic stage if determinable.

Both in clinical and life insurance activities there appears to be considerable lack of uniformity in technique as well as in interpretation.

This lack of uniformity has resulted in a selection which would furnish material for future mortality studies that would be definitely lacking in homogeneity.

- B. The exclusion of suspected diabetics did not present the same difficulties in selection as the inclusion on some basis of those in the prediabetic stage.
- C. It appears that owing to sugar tolerance tests and improved methods of urinalysis we have been able, as stated, to exclude the frank diabetic. At the same time we have been

impressed with the fact that the mortality is greater in groups entering after middle life, and even then, is deferred because it is due to degenerative conditions which operate slowly. At the younger ages where we are often dealing with a more fulminating type of the disease, we can more readily exclude the undesirable risks.

We have purposely omitted causes of death in this paper and deductions therefrom as to correlative conditions.

During this study the following points were also brought out as, at least, worthy of consideration:

1. The necessity for a uniform sugar tolerance test properly controlled.
2. The desirability of sugar tolerance tests as a matter of routine over certain ages and for certain amounts, possibly even below \$10,000, where there is a record of sugar.
3. The desirability of including the one-half hour observation after glucose in a sugar tolerance test with at least observations at fasting, one, two and three hours from the beginning; that is, five in all.
4. The desirability of companies employing identical laboratory technique.
5. A normal fasting blood sugar observation had in itself no conclusive significance and it does not rule out diabetes.
6. Concurrent history of cholecystitis.
7. The necessity in prognosis of duly considering the incidence and amount of glycosuria as well as sugar tolerance by blood chemistry.
8. The ultimate nature of the mortality rather than the immediate, in cases not frankly diabetic.
9. The concurrent association of cardio vascular conditions in diabetics which was greater in mild diabetes of long duration than in severe diabetes of short duration.
10. The fallacy of accepting a single observation two and one-half hours after the administration of glucose in which some real diabetics would be missed, and that a normal

fasting blood sugar level and a normal level three hours after glucose does not rule out diabetes.

11. The fact that a diagnosis of diabetes cannot depend entirely upon blood sugar observations.
12. The probability that two blood sugar determinations are possibly misleading, the ideal being five, viz., fasting, one-half hour, one, two and three hours after glucose.
13. That in diagnosis, short-cuts are likely to be productive of fallacious conclusions.
14. The presence of a prolonged high sugar tolerance curve with or without glycosuria is indicative of potential if not frank diabetes.
15. The transition from suspected or potential diabetes into that of frank diabetes mellitus is gradual.
16. Urinary sugar is important in diagnosis, both in respect to incidence and amount.
17. A familial history of diabetes was of noticeable frequency.
18. That a half hour or one hour blood sugar determination in the course of a sugar tolerance test, if it shows a distinct rise, is indicative in specimens received from a distance of efficient preservation and that glycolysis has not taken place.

Recent clinical studies define Renal Diabetes in such a way as to restrict the inclusion in that category very materially. Whatever the definition, the fact remains that there are a considerable number of glycosurics who may be classified as neither frank diabetics nor renal glycosurics and whatever they may be called, pre-diabetics, potential diabetics, or what not, they are insurable on some substandard plan.

From the study above described we are committed to the view, at this juncture, that, from the life insurance angle, glycosuria must be considered as a basic phenomenon from which we start fundamentally in the differential diagnosis of diabetes mellitus whether we are prepared to affirm or deny that they are synonymous terms.

At any rate we must be alive, from the life insurance standpoint to the fact that, with the exception of the so-called harmless renal type, the progress from an apparently occasional glycosuria to a graver type, even though it may be extremely slow, is probably a more common occurrence than we have heretofore believed. We should also realize that possibly we proceed from one to the other by insensible gradations and that between them there is no sharp line of demarcation, the difference being one of degree and not of kind.

As a result also of this study we suggest the possibility of the use of some uniform formula—not necessarily the one above outlined—expressive of mortality values likely to be experienced in order to record more precisely our prognostic efforts based on criteria of accepted importance.

DR. CRAGIN—I want to congratulate Dr. Brathwaite on his very ingenious method of selection and the results which have been obtained at his hands over a period of years. As I understand it, he has been collecting data since 1920, with the revision for blood sugar analysis since 1925.

Our company experience is based on data obtained since 1927 and traced to anniversaries in 1932. Originally our work was purely on urinary findings after a test meal but on June 1, 1927, we started in with the sugar tolerance test, making a single fasting blood test and a postprandial test at the end of $1\frac{1}{2}$ hours. We first used the Folin-Wu Micro method and afterwards went on the Folin Micro method. We continued the test meals with the sugar tolerance test for a while but this did not prove satisfactory and we went on to 100 grams of glucose which was cut down to 50 grams of glucose about 1929. These are the only changes we have made in our procedure.

We have had the actuaries take off some figures of our mortality experience on our sugar cases from the years of issue 1927 to 1931, traced to anniversaries in 1932, according to the compilation in Dr. Brathwaite's paper:

MORTALITY EXPERIENCE ON CASES ON WHICH A
BLOOD SUGAR DETERMINATION HAS BEEN MADE

Years of Issue 1927-1931 Traced to Anniversaries in 1932

Standard Experience by Lives

706 Entrants

| Issue Ages | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|---------------|-------------------|--------------------|------------------|-------|
| To 39 | 672 | 1.53 | | |
| 40-49 | 393 | 1.89 | 2 | 105.8 |
| 50 & Over | 326 | 3.92 | 3 | 76.5 |
| Total | 1,391 | 7.34 | 5 | 68.1 |

| Policy Years | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|-----------------|-------------------|--------------------|------------------|-------|
| 1 | 706 | 2.84 | | |
| 2 | 326 | 1.91 | | |
| 3-4 | 331 | 2.33 | 5 | 214.6 |
| 5 | 28 | .26 | | |
| Total | 1,391 | 7.34 | 5 | 68.1 |

Cause of Death: 4th year—Cardiac failure
Suicide
3rd year—Coronary Thrombosis
Acute Myocarditis
Heart Block

Substandard Experience by Lives

221 Entrants

| Issue Ages | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|---------------|-------------------|--------------------|------------------|-------|
| To 39 | 172 | .38 | 1 | 263.2 |
| 40-49 | 173 | .85 | 2 | 235.3 |
| 50 & Over | 108 | 1.30 | 2 | 153.8 |
| Total | 453 | 2.53 | 5 | 197.6 |

| Policy Years | Exposure Years | Expected Deaths | Actual Deaths | Ratio |
|-----------------|-------------------|--------------------|------------------|-------|
| 1 | 221 | .92 | 2 | 217.4 |
| 2 | 121 | .76 | 2 | 263.2 |
| 3-4 | 99 | .74 | 1 | 135.1 |
| 5 | 12 | .11 | | |
| Total | 453 | 2.53 | 5 | 197.6 |

Cause of Death: 2nd year—Suicide
3rd year—Diabetes
1st year—Carcinoma-Rectum
2nd year—Accidental Burns
1st year—Cerebral Hemorrhage

Included in the above are cases that have shown sugar on examination, cases with a history of sugar at some time in the past and some with a family history of one or more cases of diabetes.

The expected deaths have been figured by a basic mortality table representing the standard experience on all cases from 1920 to 1926 observed to the 1927 anniversaries of companies contributing to the Joint Occupation Study of 1928.

I had these made up according to the same manner in which Dr. Brathwaite studied his cases for comparison. Of course, our figures are much smaller than his.

I may say that up to the first day of January, 1933, we had requested 4,196 sugar tolerance tests. Of these, 2,628 were completed, leaving 1,568 cases in which we had no response and I shall have to leave it to your own imagination to know how many of these were true diabetics who were certain they could not stand this test. Of the completed cases, 21 were reinstatements not approved, 255 were not taken, 739 were declined, 92 issued and terminated the same year, 130 sugar combined with other codes, and 23 had insufficient quantity.

Along the first of our underwriting there was noted a certain fear of our test not wholly due to the fear of not passing it. This has been largely overcome and we have found it easier to get the test since we moved on to the 50 grams of glucose.

I would like to call your attention to a statement made by Dr. Folin at the Medical Directors Meeting in 1928 in which he said: "I think all of us medical school fellows are now pretty well agreed that at all events the sugar from non-diabetic urines is to the extent of at least 50% something else than glucose." We have been checking up on our positives to make sure that it is glucose that is causing the reaction.

We have made two separate investigations recently with the sugar tolerance tests. The first one was the applicants in which there was a family history of one or more cases of diabetes.

We sent our 348 requests. 222 responded and 42 tests had not been returned when these figures were made up. Of these 222 returned, 144 were given standard insurance, 17 were rated on the amount of urinary sugar found although the blood sugar was within our limit of 160 mgs., and 38 were rejected—a total of 55 were rated or rejected on the sugar tolerance test. Twenty-three others in this group were rated or rejected for other causes which had nothing to do with the sugar tolerance test. In this series of 222 cases, 59 had sugar above the clinical normal of 120 mgs. and there were quite a number which were borderline—150-160 mgs. Translated into terms of dollars and cents, the total amount of business represented was \$1,547,700. Of this, \$942,700 was issued at standard rates, \$89,000 was rated, \$286,500 was rejected outright on account of the sugar tolerance test, we feeling certain that they were true diabetics. The rest of the money went into the rated or rejected cases for reasons other than sugar tolerance. As you will see by the analysis of the above figures, the percentage of true diabetics in cases of family history is high enough to warrant serious consideration.

In order to have some check against these figures we took 100 of our employees in the office in which there was definitely no family history of diabetes and gave them our sugar tolerance test. In none of these cases did we find a diabetic. One case had a postprandial at the end of $1\frac{1}{2}$ hours of 154 mgs. but his urinary sugar was —100 mgs. It is interesting to note that one smart boy in the organization wanted to get an examination free as he had had a family history of diabetes and came in, telling us that his family history was negative. We found him to be a true diabetic. His postprandial glucose was 182, he had 1% of sugar in the urine, and when we confronted him with the facts he confessed the truth, so he was thrown out of the series. The fasting average in these cases was 95 mgs. per 100 c.c., the postprandial average was 89 mgs. per 100 c.c., the glycosuria average was 132 mgs. per 100 c.c. We had all races: American, Irish, Italian, English, German-American, Lithuanian, French, Polish, Swede, and Scotch.

We call for a sugar tolerance test now:

1. Where there are two or more cases of diabetes in the family history.
2. Where there is one case combined with overweight in the applicant.
3. Excessive overweight.
4. An insurance record of sugar—whether Q'd or not (and I may say in this connection that we have picked up quite a number of diabetics by this procedure).
5. Sugar on examination, except gross amounts of sugar.
6. History of rapid loss of weight with slow return.
7. Urinary findings in the Home Office of low specific gravity with high glycosuria, around 300 mgs.

In regard to the conclusions which Dr. Brathwaite makes, it would be a mighty fine idea for us to get together and have a uniform sugar tolerance test, properly controlled, and we are willing to go along with the majority opinion.

The question of the desirability of sugar tolerance tests as a matter of routine is one that will probably get a considerable reaction from the Agency Department. However, it might be worth while.

We should employ identical laboratory technique although we have noticed a growing tendency recently to change from the ordinary methods of sugar examination to the Micro technique.

We agree that normal fasting blood sugar observation has no conclusive significance and does not rule out diabetes.

The seventh conclusion is one that has been considered in our underwriting and on the following page is given the blood sugar investigation covering the amounts, for what they are worth.

We are very anxious to see what happens to the renal glycosurias. The clinicians all tell us that renal glycosuria is not a dangerous affair. We wish we could get some figures on the ultimate mortality.

The other conclusions have been either worked into this discussion or else are debatable points.

BLOOD SUGAR INVESTIGATION
1927-1930 (Traced to Anniversaries in 1931)
Time $1\frac{1}{4}$ — $1\frac{3}{4}$ hours

| P.P. | No. | Exposed | Actual Loss | Expected A. M. Select | % | Cause of Death |
|-------------------------------------|------|-------------|-------------|--------------------------|-------|-------------------------|
| 120 mgs. or less | | | | | | |
| | No. | 484 | 1 | 2.9 | 34.5 | Cancer |
| | Amt. | \$4,867,960 | \$10,000 | \$30,059 | 33.3 | |
| 121-140 mgs. | | | | | | |
| | No. | 480 | 3 | 3.8 | 78.9 | { 1 Acc. Gas Poisoning |
| | Amt. | 3,744,264 | 17,000 | 30,965 | 54.9 | { 1 Acc. Burns |
| | | | | | | { 1 Coronary Thrombosis |
| 141-160 mgs. | | | | | | |
| | No. | 275 | 3 | 2.3 | 130.4 | { 1 Myocarditis |
| | Amt. | 2,289,448 | 25,000 | 18,744 | 133.4 | { 1 Suicide (2 pols.) |
| Over 160 mgs. | | | | | | |
| | No. | 58 | — | .9 | | |
| | Amt. | 421,000 | — | 5,020 | | |
| Time less than $1\frac{1}{4}$ hours | | Issued | Actual Loss | | | Diabetes |
| | | 30 | 1 | | | |
| | | \$177,000 | \$1,000 | | | |
| More than $1\frac{1}{4}$ hours | | | | | | Cerebral Hemorrhage |
| | | 75 | 1 | | | |
| | | 953,000 | 5,000 | | | |

AFTERNOON SESSION

DR. SCADDING—This afternoon we have with us a most distinguished guest in the person of Dr. Lewellys F. Barker, Professor Emeritus of Johns Hopkins. He is to address us upon a subject which I know is of vital importance to us all in the selection of a class of cases which has given us much perplexity. It is not difficult to introduce Dr. Barker. The chief characteristic of such great medical men as William Osler, Lewellys Barker, Alvah Gordon, only to mention sons of Canada, is, I think, that they possess in the highest degree that rare gift of the gods, "the lifting hand".

In introducing the members of this organization to you, Dr. Barker, permit me to say that they represent in some way or at some time all the branches of the art of medicine, and are more or less clinically minded. They adore "the angel of life insurance" and, certainly more than any other class of medical men, have fathomed to some degree that abyss, the mind of the actuary. Their aim is to serve their companies faithfully and, with the actuaries' aid, to continue to make worthy contributions to general medicine.

DR. BARKER—Your President, Dr. Scadding, has been very flattering in his introduction of me but the members of this audience are exceptionally able to recognize "what a man's life is worth" and will readily discount any over estimate that he may have made. Over forty years ago when I was in the medical school, Dr. Scadding and Dr. John Caven were internes in the Toronto General Hospital. I shall never forget the admiration that they inspired in us then and it is no surprise to me that he has ultimately attained to the high position he now holds.

When he invited me to address you I felt highly honored, and if my paper excites some discussion I shall be very pleased.

ON THE CONSIDERATION OF EVIDENCES
OF NERVOUS OR MENTAL DISEASE (OR
OF PREDISPOSITION THERETO)
IN THE SELECTION OF
INSURANCE RISKS

By LEWELLYS F. BARKER, M. D.
Baltimore, Maryland

The importance of the recognition through a study of the familial and the personal history of any especial predisposition to nervous or mental disease, on the one hand, as well as, on the other hand, the necessity of the detection by physical and mental examination of early signs of neuropsychiatric disease already existent in applicants for life insurance, would seem to need no emphasis to members of this audience. As medical directors of great life insurance companies, you have doubtless been impressed with the number of insured persons who have developed organic or severe functional nervous disorders, or who have manifested psychoses that required institutional treatment relatively early in life. The influence of these maladies upon the morbidity rate and upon the mortality rate among the insured must not only have caused you some concern but also have excited your interest in devising ways and means whereby better estimates than hitherto can be made of the chances of their development in single persons when they apply for insurance.

With the rapid increase of knowledge characteristic of our time, it should be possible to assign to the classes of standard and of various substandard risks more accurately than ever before the applicants among whom the suspicion of the danger of neurological or psychopathological development has become aroused.

THE HEREDO-FAMILIAL ORGANIC NERVOUS DISORDERS

Neurologists have made us familiar with a whole series of organic nervous maladies in which inheritance plays an im-

portant part. Thus certain of the progressive muscular atrophies, the muscular dystrophies, the spastic spinal paralyses, Friedreich's ataxia, Marie's cerebellar heredo-ataxia, Huntington's hereditary degenerative chorea, Thomsen's myotonia, familial periodic paralysis, familial myoclonia, and amaurotic family idiocy are described in the textbooks as diseases that occur predominantly in certain families, owing to hereditary transmission. Though some of these maladies if they are to occur in a given person will make their appearance so early that insurance examiners will have no concern with them, there are others that appear later in life after an apparently healthy childhood and early adulthood. It is the latter group of course for which the insurance examiner must be on guard through very careful questionnaires concerning the family history that will reveal their presence or absence among blood-relatives of applicants.

In addition to these more strictly neurological diseases, it must be remembered that certain vascular diseases that endanger the central nervous system also tend to run in families. I refer especially to the atherosclerotic maladies that may cause either sudden death or severe disability through the occurrence of cerebral apoplexy or cerebral thrombosis. An analysis of family histories quickly teaches us that there is a definite tendency in certain families to die at approximately the same age as a result of such vascular accidents involving the brain. In other familial groups, an atherosclerotic malady may tend to involve especially the renal arterioles and lead ultimately to death from uræmic intoxication of the brain that causes convulsions or coma.

THE HEREDO-FAMILIAL MENTAL DISORDERS (PSYCHOSES AND PSYCHONEUROSES)

Though diseases as such are not directly inherited, the disposition to develop certain psychoses and psychoneuroses undoubtedly has its basis in the genotype. This seems to be true of the manic-depressive psychoses, of schizophrenia, of idiopathic epilepsy, of hysteria, and of neurasthenia.

That the *manic-depressive psychoses* run in families seems definitely to have been established and attempts have been made

to explain the kind of inherited disposition upon a Mendelian formula. Certainly when mania, melancholia or circular insanity has occurred in the family of an applicant for insurance, the fact should be carefully weighed in estimating the expectancy group to which the applicant probably belongs. It is interesting that a special type of physical habitus, namely the so-called apoplectic or pyknic habitus, seems to be associated with the disposition to develop psychoses that belong to the manic-depressive group. Persons of pyknic habitus tend to be rather short and thick set, to have round faces, short necks, and short fingers and toes, to show signs of obesity and not infrequently early development of high blood pressure.

The *schizophrenic mental disorders* (including so-called dementia præcox) tend also to run in certain families. The inherited predisposition thereto seems to conform to a somewhat peculiar recessive Mendelian formula. The environmental factors that contribute to the development of the schizophrenic reactions are being carefully studied, though as yet these are not well understood. If the family history of an applicant for insurance reveals the existence of relatives who have exhibited schizophrenic symptoms, the applicant himself should be most carefully examined for the presence or absence of evidence of so-called "autistic behavior" as well as for "defects of capacity for normal emotional responses". The physical habitus of those predisposed to schizophrenic anomalies is most often of asthenic rather than of pyknic type; it is the tall slender person with long face, long extremities, long fingers and toes, thorax paralyticus, tendency to emaciation and to low blood pressure that should make one keep in mind a possible predisposition to tuberculosis on the one hand and to schizoid temperaments on the other.

Epileptic syndromes though often the result of acquired brain injuries are sometimes met with as expressions of a special inherited reaction-tendency that characterizes certain families. In members of such families the so-called "stigmata of degeneration" are frequently observable; in some of them, too, endocrine anomalies (hypoparathyroid, hypogenital) are encountered.

That *psychopathic personalities and psychoneurotic manifestations* also tend to be much more common in some families than in others is matter of common knowledge. In recording the family history of an insurance applicant, therefore, as much information as possible should be given regarding the existence of psychopaths and of neurotics among the relatives.

ACQUIRED NERVOUS AND MENTAL DISORDERS

In the *personal history* of an insurance applicant should be registered all nervous and mental disorders that have actually been experienced by the candidate during his lifetime. Some of them, say a few convulsions in childhood, or a poliomyelitis with residual monoplegia may be of no great significance for life expectancy, but others of them, for example, a lethargic encephalitis with residuals, a cerebrospinal syphilis, a tabes dorsalis, a dementia paralytica, a subacute combined sclerosis, a disseminated sclerosis, a vascular lesion of the brain or spinal cord, or a tumor of the nervous system may definitely exclude the possibility of insurance, or if insurable at all will relegate the candidate to a substandard class.

THE PHYSICAL EXAMINATION OF THE NERVOUS SYSTEM OF AN APPLICANT

Unfortunately, the personal history and family history given by an applicant for insurance is not always wholly reliable either from inadvertence, from ignorance, or in some instances from wilful intent to deceive. It is therefore important that a thorough physical examination be made and that this should include an examination of the central and peripheral nervous functions. Every general medical practitioner, thanks to the training now given in our medical schools, should be able to make the kind of neurological examination that will detect any marked disturbance (1) of general sensibility and of the special senses, (2) of motility and the state of nutrition and tonus of the musculature, (3) of the deep and superficial reflexes, (4) of co-ordination and (5) of the higher brain functions of speech,

writing, conduct and behavior; he should discover too any pupillary anomalies, any important changes in the eye-grounds, and any striking disturbances in the domain of the intellect and the emotions. Even if he be not versed in all the more refined methods of diagnosis, he should be sufficiently master of general diagnostic methods to know when to be suspicious and when to suggest the aid of specialists in neurology or psychiatry because of his doubt. No insurance company would want a medical examiner who would fail to detect the presence of anisocoria, of an Argyll-Robertson pupil, of a choked disc, of a Parkinsonian facies, of fibrillary twitching of the thenar and hypothenar eminences, of absent knee jerks or ankle jerks, or of a positive Babinski reaction. Nor would a company today be satisfied with an examiner who failed to notice a dysarthria, an inability to add, subtract, and multiply, a marked psychomotor acceleration or retardation, a tendency to disorientation as to time and place, or striking eccentricities of behavior.

THE DANGERS OF APOPLEXY AND OF SUICIDE AMONG THE INSURED

I wish next to speak of two especial causes of death among insured persons—apoplexy and suicide.

In persons over 50, *death by apoplexy* (cerebral hemorrhage) is by no means uncommon. It is usually preceded by a longer or shorter period during which high blood pressure and signs of arteriosclerosis have been demonstrable. As a help in the selection of insurance risks, medical directors should lay great stress upon the importance of reports by their medical examiners first, upon the family history of the applicant as regards blood pressure and arteriosclerotic changes, and, secondly, upon the physical examination for the recognition of incipient atherosclerotic changes (diastolic blood pressure above 90 with diminished pulse pressure, palpable radial and temporal arteries, and premature corneal arcus senilis); if any of these be observed, and especially if there be any history of vertiginous attacks or of matutinal headaches the examiner's suspicions should be aroused.

There is no sure way of anticipating *death from suicide*, though here again there are certain criteria that may well arouse concern and may possibly make it wise to place the applicant if accepted at all in a substandard group.

Our knowledge of the conditions of suicide has made marked growth in recent decades during which there has been a definite increase in the suicide rate in all civilized countries. It is said that in France the rate has quadrupled during the past century, and that among German races the tendency to suicide has been especially marked (even child-suicide having been on the increase) whereas among Slav populations suicides are less common. Besides ethnic factors, religious and philosophic influences play a part; thus among those who believe in God and a life after death suicide is less common than among atheists; and, among believers, suicides are more common among Protestants and Jews than among Roman Catholics. Cultural relations also play a role, since suicides are least numerous among primitive and ignorant peoples and most common among nationalities that have reached a high grade of culture and refinement. More suicides occur in fine sunny weather than during cold cloudy wet days or months; the suicide rate tends to increase from the beginning of the year until it reaches a maximum in late spring or early summer after which it decreases slowly until the end of the year. During economic crises there may be a great rise in the suicide rate; I have been impressed with the numbers of suicides reported in our daily press during the past year. On the other hand, after great catastrophies like wars or destructive earthquakes, the local suicide figures fall. More men than women commit suicide, but the rate among women has been growing with the changing conditions that make the life of women in industry and in society approach more nearly to that of man.

The majority of suicides occur among persons of neuropathic or psychopathic constitution; as a rule they follow sudden impulses (rather than well-defined motives) in persons of marked emotional instability. Some of the persons were earlier of strong character, but we fear suicide even more among those of weaker character who come under prolonged stress and strain. Evidence

of family tendency to suicide is abundant. For example, a wealthy neuropath whom I saw first six years ago committed suicide last spring and on reviewing the family history I have found that his brother had committed suicide four years earlier; both had exhibited sex conflicts and behavior difficulties, both had been alcoholic, their father had been an excessive drinker, and their mother had suffered from a so-called nervous breakdown!

In insurance work, because of the danger of suicide, it would seem that evidences of neuropathic or psychopathic heredity, of family history of suicide, of personal emotional instability, of sex conflicts, of alcoholic tendency, of self-depreciative temperament, of feeble character, of abnormal religious and philosophic views, and of predisposing ethnic origin in candidates should be given due consideration when making decisions as to risks assumed.

PROGNOSIS FOR THE INDIVIDUAL AND FOR THE GROUP IN WHICH HE FALLS

The examination of the single person from the neuropsychiatric standpoint may give clues that are valuable for estimating the especial life dangers to which he is exposed but it is most important to the life insurance medical director who is to decide upon the group of selected risks in which he falls in case his application is not rejected. Those of us who practice medicine are most interested in consideration of the prospects for the health and for the duration of life of each single person that we study and treat; but you who decide upon the policies of life insurance companies as regards groups of substandard risks are more directly concerned with the death rate within the mass that corresponds to each group that you set up, since the financial safety of the insurance company depends, as I understand it, upon fairly accurate anticipation of the rate of mortality for each of your groups. Thanks to the studies of the statistics of the actual experience of many companies with regard to mass mortality as a whole and to the mortality of each of several groups of selected risks, it is possible to make insurance contracts that are reasonably fair to both insurer and insured. Prog-

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ress toward greater accuracy is, however, being made all the time through better medical examinations of single candidates, through ever better judgment upon the part of medical directors as to the group to which a single candidate should be assigned, and through ever more accurate statistical and actuarial studies.

On looking over, recently, the Medical Impairment Ratings of the Actuarial Society of America, my impression was that the ratings were over liberal to candidates with history of certain nervous disorders.

If what I have said should be of any help in the weighing of evidence regarding the disposition to neuropsychiatric diseases that may have a bearing upon life expectancy I shall be pleased and feel well repaid for the preparation of this brief paper. And if the points developed should do something to help on life extension campaigns among those that are insured I shall be all the more glad.

DR. GROSVENOR—Dr. Barker has presented the factors in connection with the importance of familial and personal history in the selection of applicants for life insurance with a predisposition to neuropsychiatric disease in a very clear and helpful manner.

We are all very much interested in the increased mortality and morbidity attributable to these diseases.

The family and personal history of the applicant is as important to the medical director as it is to the clinician who endeavours to interpret the constitutional tendencies of his patient and to discover, if possible, any inherent developmental defects or stigma, which may point to the probability of abnormal manifestations in the future. It is not always possible to obtain a satisfactory history, even with painstaking effort, because of several reasons as Dr. Barker has mentioned.

When nervous or mental disease is present, it is not usual to encounter any great difficulty in arriving at a general diagnosis but in life insurance examinations, the applicant is apparently well or at most borderline, thus requiring a great deal of acumen to discover any early manifestations of abnormality. It would be helpful if the medical examiner would observe and record any symptoms which would be indicative of a constitutional neuro-

mental inadequacy such as, easy fatigability, morbid introspection, vaso-motor or emotional instability.

As Dr. Barker has indicated, etiologically, nervous and mental diseases have for their background an inherent biological instability. These deep rooted defects may always remain recessive but an individual who has an associated organic disease, or who is faced by mental conflict and stresses, may be predisposed to a neuropsychosis.

In discussing causative factors, Dr. Barker mentions cardiovascular changes that affect the nervous system. It is apparent that cardiovascular and nervous diseases do show that there is a tendency in certain families to die at about the same age.

In this connection it may also be stated that sex, nationality, occupation and habits, together with previous history of disease, are factors of importance in evaluating our selection. Some diseases are found more particularly in certain age periods, thus in early adult life cerebral abscess and disseminated sclerosis may show a greater incidence just as acute rheumatism, endocarditis, tuberculosis, gastric and duodenal ulcer are more apt to occur during this period.

In adult life, nervous and cardiovascular changes are prone to be manifest due to physiological changes and mental stresses. It is believed that such diseases appearing at this time of life are more resistant to treatment and hence, the prognosis is more grave and disability prolonged. The psychoneuroses are probably more likely to be associated with sedentary occupations among those doing mental work.

The whole field of pathological changes incident to diseases with inherent biological instability is very complex and opens a field for intensive research. At the present time there is no complete answer to this problem. Just how an individual will react to the conditions of his environment we do not know. Undoubtedly many individuals in the face of inherent defects are able to adjust themselves while others fail. Although predisposition to neuropsychiatric disease is essentially constitutional, a predisposition may be acquired by exogenous or endogenous toxins, trauma or bodily disease.

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The selection of lives for life insurance purposes requires judgment in order to focus the facts as secured by the medical examiner. If our examiners possess the clinical faculty to detect abnormal family and personal history, including any stigmata that would render the applicant more liable to the development of neuropsychiatric disease, it is a relatively simple matter to correlate the facts and place the applicant in his proper group. Finally, if our selection is based in accordance with the lines suggested by Dr. Barker, many cases showing a trend toward nervous and mental disease can be detected and action taken in accordance with the facts presented.

DR. SCADDING—I would like to ask a question or two that occurred to me while Dr. Barker was reading his paper. Has he found that arcus senilis is an important prognostic sign? Is it sometimes an hereditary affair? I remember Marcus Gunn, renowned in London, in my student days, saying he thought arcus senilis of no prognostic significance. In practice, some time after that, I ran across one or two cases of families wherein the arcus senilis was found to be a marked sign. One case I recall very well: the father of the family was a confirmed alcoholic. He had a very marked arcus senilis, as had also his wife who was a total abstainer. There were four children in the family. They are all living today and are all over sixty and they all exhibited arcus senilis before their twenty-first birthday.

I am going to ask Dr. Barker if he would allow us to add the word Roman to his reference to the Catholics. As a Catholic, perhaps a so-called Protestant, I would like to express my profound respect for the discipline of the Roman Catholic Church. As a Medical Director, I venture to say if we could get all our applicants to join that Church, our losses from suicide would be negligible.

DR. DALEY—I really think that the diffidence that this Association is showing in regard to the discussion of Dr. Barker's paper is because of our feeling of helplessness when it comes to the handling of neuro-psychological problems. They are very difficult things to handle. I can remember years ago, when I

was associated with Dr. Dana and Dr. Frankel, examining a case with Dr. Frankel. He said, "Well, we have examined you very thoroughly. We have heard the complaints. We believe they are real. We do not know what is the matter with you, so we say you have neurasthenia".

Nervous breakdowns are the cause of considerable expense to all insurance companies from their disability viewpoint. The great trouble in examining cases is that the nervous side of them is not brought out. It is very difficult, indeed, in examining a case that shows some nervous abnormality to follow that up. It takes a trained mind to do it and you must take plenty of time to do it. What this Association needs more than anything else is a line of guidance to segregate at the initial examinations those cases which should be studied further from a neuro-psychological point of view. I know since we have had a neurologist on our staff, he has been a great relief to us in handling cases of nervous depression, neurasthenias, nervous breakdown and such.

DR. MCCRUDDEN—This morning I referred to the abnormally high proportion of suicide losses among glycosuria subjects who were insured after passing a satisfactory blood sugar tolerance test. This group must include many whose glycosuria was emotional. Was the emotional glycosuria an expression of the nervous instability which ended in suicide?

DR. OLD—I would like to ask Dr. Barker if he finds a psychoneurosis ever develops later on into a psychosis. I don't know if it is the experience of you other gentlemen, but it seems to me that the neurologist as a rule, certainly with applicants we have for life insurance, never goes beyond the diagnosis of psychoneurosis. I have yet to see a letter written with regard to nervous breakdown in the past in which the answer didn't always come back as a case of psychoneurosis. I don't believe we have ever received a report with a diagnosis of psychosis.

DR. SCADDING—Dr. Barker, do you wish to say something further?

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DR. BARKER—I am very much gratified that the paper should have aroused so much discussion. I have been interested in all that has been said and am particularly pleased to know that Dr. Grosvenor and his group approve of the main points brought out in the paper. He is quite right in emphasizing the importance of age groups in diagnosis, especially with regard to multiple sclerosis. If a nerve malady begins late in life it is rarely multiple sclerosis, for this disease usually begins fairly early in life. I believe that every doctor, as a matter of routine, should test the abdominal reflexes carefully and also, having the patient recumbent with arms folded and feet held down, he should ask the patient to sit up in order to test the strength of the abdominal muscles. In this way he could rule out multiple sclerosis or become suspicious of its existence. The first signs of multiple sclerosis are, as a rule, loss of abdominal reflexes and weakness of the abdominal muscles. If the abdominal reflexes are active and the patient can sit up with his arms folded across his chest, in ninety-nine times out of a hundred, one can rule out multiple sclerosis.

Dr. Scadding has spoken of arcus senilis. I think that the exception he takes to the evaluation of that symptom is well taken. Of course, arcus senilis is an evidence of degeneration of the cells in the periphery of the cornea. We all know how common this is in arteriosclerotic patients in advanced life. I mentioned it mainly because it is so common among arteriosclerotics. I think it is true that certain families must inherit diminished resistance of the periphery of the cornea or a poor local vascular supply, and so develop arcus senilis early.

Dr. Daley spoke of the relation of neurasthenia to so-called nervous breakdowns or psychoneuroses. So did Dr. Old. Well, the terms "neurasthenia" and "psychoneurosis" are somewhat abused. Physicians dislike very much to use the terms "psychosis" and "psychoses". But in every psychoneurosis, there is some disturbance of the mental state as well as of the physical state. Psychoneuroses are psychopathies to a certain extent, though they do not fall within what we ordinarily call "psychopathy".

The business man, under great stress and strain for months, who has had no holidays and hasn't slept at night, will become fatigued and a little irritable and will be below par for a while. We say that he has a "reactive psychoneurosis", or a neurasthenic state, but he ought quickly to make a good recovery if the cause is removed. There are also true hystericals. Hysteria is a definite malady of the nervous system. Then there is the "chronic invalid reaction" that we see so frequently; the patient is never well but is always complaining. That is due to constitutional make up as a rule. We call the condition that is manifested the "chronic invalid reaction". A few of the patients under the best care may get well.

The "psychasthenic states" are perhaps most often misunderstood by the general practitioner. Sometimes these patients have no outspoken obsessions but exhibit phobias, marked indecision and vacillation, over-conscientiousness, and fear of doing something wrong. Often they are unwilling to do the things of every day for fear of making mistakes. We see the severer cases in hospitals and in psychiatric institutions. Psychasthenics can best be handled by a man who understands them. A family doctor can help a great deal if he will tell the patient that there is no foundation for his fears, will help him to overcome his over-conscientiousness, and urge him not to vacillate, for it is often better to make a decision and make a mistake than to leave a matter long undecided. The psychasthenic should be told that he can do the thing he fears he cannot do, and that the thing he fears will happen probably will not happen! Forcible statements from a good, sensible family physician will greatly help to carry a person of psychasthenic constitution along.

One thing I should like to emphasize especially in connection with "neurasthenias" and "nervous breakdowns". A great many of the patients do not belong in the neurasthenic group at all. They are not ordinary psychoneuroses at all. The severer "nervous breakdowns", in the majority of cases, are in my opinion mild psychoses. Many of them are "true depressions" that last a year or a year and a half no matter what you do for them. The patients lose interest in things, complain of lack of appetite,

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and of feeling unequal to the demands of life; they feel sad, blue and gloomy and are prone to withdraw from social groups. They give their families a great deal of trouble. Suicidal ideas may emerge and some of them do commit suicide.

Some of these patients have such bad "nervous breakdowns" that they have to take a "rest cure" and are put in a sanatorium, a nursing home or a private ward of a hospital. They are passing, in reality, through a melancholic or depressive phase of a mild manic-depressive psychosis that has to run its course. The average duration is a year to a year and a half, no matter where you treat the patient and no matter how good the psychiatrist or neurologist who has him in charge. The physician can help carry the patient along, help him over hard places, reassure him and keep him from doing the wrong thing. I don't know any sure way of shortening a true "depression"; I do not think that any psychiatrist does, though if the patient will give himself over to the best treatment early, I have gained the impression that we may shorten the duration some.

After the stage of "melancholy" is over, the patient may become a little "too well", that is to say, he may be slightly "elated", and exhibit "psychomotor acceleration". Everything is easy for him then; nothing is difficult. The world is his oyster. These patients tend to oscillate between what they call "nervous breakdown" (depressive phase) and what they call "being well", when they are really "high" (elated phase). When they are "high", they talk all the time, jabbering away constantly and too quickly associating one subject with another that is not very pertinent to it.

Familiarity with the mild depressive phases and the mild elative phases in patients of manic-depressive constitution seems to me to be very important, and I wish that every general practitioner had enough psychiatric training and insight to recognize these conditions when he meets them. If such conditions were better understood, we might prevent many suicides.

DR. SCADDING—The next paper on our program will be presented by Dr. William B. Bartlett.

PNEUMOTHORAX

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Pneumothorax is the name applied to the presence of atmospheric air or gas in the pleural cavity. The condition was first recognized by Riolan, a contemporary of Harvey, and was first described by Itard—a French physician who coined the word “pneumothorax” in 1803. His studies were made post-mortem. The disease was first recognized during life by Læneç in 1819. His investigations, particularly of the symptoms and diagnosis were so thorough that posterity has been able to add very little to them.

The cases occur most commonly in early adult life, between twenty and thirty, corresponding to the period when pulmonary tuberculosis is most frequent. The condition is occasionally observed in infants and children. It may be unilocular or multilocular depending on pleural adhesions; partial or total; unilateral or bilateral. It may be simple or complicated by an effusion of serum, blood or pus. The condition is always secondary to an accident or a diseased condition of the lung, pleura or chest wall although the primary cause is sometimes not discoverable. The air may gain entrance to the pleura through a perforating wound of the parietal pleura and chest wall or may come from a perforation of the pulmonary pleura connecting with the lung, bronchi, trachea, cesophagus, stomach or intestines.

The commonest cause for the condition is disease of the lung; in the great majority of cases pulmonary tuberculosis. Among Biach's oft quoted 918 cases from three Vienna Hospitals 715 (77%) were attributed to this cause. It is common in early tuberculosis and results from rupture of small subpleural tubercles. Its infrequency in cases with large cavities and in the more chronic type of the disease is due to the more extensive and firmer pleural adhesions present in such cases. Other causes of

pneumothorax are bronchiectasis, abscess or gangrene of the lung, pulmonary infarct, lung tumor and echinococcus disease. It has been noted in asthma and emphysema but is rare in these conditions in older individuals. Abscess and hydatid disease of the liver, ulcer or cancer of the stomach, cancer of the œsophagus and perforation of the œsophagus by a fish bone have been recorded as causes. It may also be due to a penetrating wound of the chest wall or of the lung such as stab or gunshot wounds but it is surprising how seldom this complication arises. The operation of thoracentesis may be responsible.

Owing to the elastic tension of the lung and the consequent negative pressure in the potential pleural space the admission of air to the pleural cavity is followed by a collapse of the lung which may be partial or complete. The mechanical conditions differ somewhat, depending on whether the communication of the pleural space with the outside world is open, valvular, or closed, and depending on the size of the perforation and the presence or absence of adhesions.

Three types of pneumothorax have been described—closed, open, and valvular. In the open variety a fistula connects the pleura with the outside air either through the lung or chest wall and atmospheric pressure exists in the pleural cavity. The valvular variety is due to a valve like action of the fistula which permits ready entrance of air into the pleural cavity but prevents its easy escape. If the collapse of the lung is sudden and complete the symptoms are urgent. Slow or partial collapse may occur without symptoms and be discovered accidentally on X-ray or physical examination.

Pneumothorax secondary to obvious pulmonary disease does not interest us from the insurance standpoint as such cases are not insurable. There is, however, a form of pneumothorax in which no definite etiological agent can be discovered. It makes its appearance without any demonstrable cause in healthy persons, in whom no signs of tuberculosis may be found either by auscultation, roentgen examination or bacteriological examination of the sputum. The symptoms are usually mild; no complications arise and complete recovery is the rule. Hamman¹ of Baltimore calls

this Spontaneous Pneumothorax and defines the term as follows: "A pneumothorax coming on in an apparently healthy individual without ascribable cause, resulting in no infection of the pleura and therefore unaccompanied by constitutional symptoms, healing rapidly and completely in a few weeks."

Fussell and Riesman² of Philadelphia collected 56 such cases from the literature in 1902 and many more have been reported since. In fact the condition is probably quite frequent and often unrecognized. Much has been written as to the probable cause. Hamman believes it arises from the rupture of superficial emphysematous blebs or from a tear of the pulmonary pleura resulting from the pull of an adhesion. To support this view may be cited the fact that many cases follow some unusual exertion or may occur after laughing, crying, coughing, sneezing or yawning. Several theories have been advanced as to the source of the superficial emphysematous blebs which are ascribed as the cause. Most Clinicians believe these blebs result from an old inactive tuberculous process. Others believe they are the result of a localized emphysema. The condition is seldom fatal and few necropsys have been reported. One reported by Meyer³ in 1917 showed an extreme degree of emphysema with many thin walled air spaces throughout both lungs. The patient was a woman in the early twenties; the pneumothorax was bilateral and extensive. There was a history of two attacks preceding the fatal one. There was no evidence of tuberculosis. Abt and Straus⁴ reported a similar case in a boy of twelve. At autopsy both lungs showed marked emphysema. The rupture was through a large emphysematous bulla.

The course of the illness in a case of spontaneous pneumothorax is entirely different from pneumothorax complicating pulmonary tuberculosis. There are few if any constitutional symptoms, and fluid, which is always present in the tuberculous variety, is seldom found in the spontaneous type. Recurrent attacks have been frequently reported. In a few cases both lungs have been involved at once and recovery has taken place. LeWald⁵ reported one case in which the collapse persisted for slightly more than a year and another in which the lung has failed to expand

after eleven years. He concludes that spontaneous pneumothorax may be present for a period of years without the development of any evidence of tuberculosis.

Many cases have been followed for years and the development of tuberculosis has only rarely been noted. A Swedish writer, Kjærgaard⁶, states in 1932 that systematic after-examination of a large number of patients with long observation periods have shown that in practice it may be taken for granted that this disease has nothing to do with active tuberculosis. He admits, however, that a frequent cause is the rupture of a scar tissue vesicle, originating from a healed tuberculous process.

The Röntgenologists are quite sure that these cases bear no relation to tuberculosis. Dr. LeWald⁷ of New York read a paper on the subject in 1925 and cited ten cases observed by him, not a single one of which gave evidence of tuberculosis. He concludes that spontaneous non-tuberculous pneumothorax may occur in a person with healthy lungs and result in complete and permanent recovery. In the discussion which followed this paper Dr. James Alexander Miller and Dr. Willy Meyer of New York, Dr. Gerald Webb of Colorado Springs and Dr. Lemon of Rochester, Minnesota, disagreed with this view and maintained that all such cases should be attributed to tuberculosis. In a recent personal communication Dr. LeWald states that he has never seen tuberculosis develop in these cases. On the other hand Dr. John B. Hawes⁸ of Boston believes the X-ray men are making a great mistake in putting out this doctrine.

Dr. Frederick T. Lord⁹ of Boston has had twelve cases under his observation. In five cases there was X-ray evidence of probable tuberculosis. In three of these there was probable calcification in the apical region and in the remaining two probable calcification at the lung roots. Of the remaining seven cases X-ray examination was negative in six. There was no record of an X-ray in one.

He writes me as follows: From this series it is reasonable to assume that spontaneous pneumothorax is due to tuberculosis in somewhat less than one-half of the cases. In this group the tuberculous process appears to be minimal and may even be

obsolete. The occurrence of spontaneous pneumothorax in patients without X-ray evidence of the disease and failure to react to tuberculin raises the question whether or not there are instances of spontaneous pneumothorax in the absence of tuberculosis. The failure of a reaction by the intracutaneous test with 1 mg. of tuberculin, as in one of my cases, may be taken to indicate the absence of an active tuberculous process, but does not disprove the possibility of rupture of a small pleural adhesion originally due to tuberculosis. The outlook in my cases is good. Eleven of twelve cases have been followed. Only one of these has died and this patient died of angina pectoris ten years after the spontaneous pneumothorax. The ten remaining cases are well after 14, 12, 11, 8, 7, 6, 3, 2 and 1 years and one after four months. There have been recurrences of spontaneous pneumothorax in three cases.

Lemon and Barnes¹⁰ in their report on fifty cases of pneumothorax observed at the Mayo Clinic state that pneumothorax furnishes strong presumptive evidence of tuberculosis. Hirschböck¹¹ made a careful study of spontaneous pneumothorax and reported several cases of his own. He states that many isolated pathological findings tend to support belief that these cases are always tuberculous. He quotes Kleenan who reported two cases that revealed tuberculosis four and seven years later.

In spite of the strong presumptive evidence that this condition is usually tuberculous in origin it remains a fact that the very great majority of these cases get well and remain well and never develop clinical tuberculosis. How should we treat them when they present themselves for insurance? Apparently the significance of a spontaneous pneumothorax is similar to that of hemothysis or idiopathic pleurisy. A careful history and thorough study is required. If tuberculosis was found to be the cause the case should be rated as such. If the case was investigated by a competent observer and no tuberculosis was revealed by physical examination or X-ray more lenient treatment is suggested. Each case must be considered on its merits but we must not forget that the condition may be due to an underlying inactive tuberculosis or be an indication of extensive emphysema and that re-

current attacks are not unusual. If the applicant is above the average weight with a good family and personal history and has been in good health for several years since the attack, he may be safely accepted.

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DR. SCADDING—The discussion will be opened by Dr. Jabez H. Elliott, Professor of History and Medicine at the University of Toronto.

DR. ELLIOTT—My first interest in the subject of pneumothorax was aroused by the paper of W. F. Hamilton in *Montreal Medical Journal* December, 1889, in which he reported twelve cases. Of these six were probably due to tuberculosis, three to empyema, one to bacillus *aerogenes capsulatus* and two occurred in the apparently healthy.

Dr. Scadding's kindly invitation to join in the discussion of Dr. Bartlett's subject was followed by an advance copy of his paper. On reading this masterly summary of the subject with its special discussion of the type of pneumothorax which will not

preclude acceptance of an applicant for insurance, I found myself fully in accord with his conclusions; so fully in accord that I felt it would be wasting your valuable time to reiterate those conclusions or attempting to present facts and opinions from the vast literature of the subject which has been so carefully studied by Dr. Bartlett in the preparation of his paper. So I concluded to look over the case records of the past five or six years at St. Michael's Hospital where I am in charge of the chest service, adding a few cases seen in consultation, and present these records to you, with the X-ray films which are fortunately available in most cases.

These cases I find fall into five classes.

1. *Traumatic*—Five cases.
2. *Induced (therapeutic)*—Not discussed.
3. *Accidental in the course of therapeutic procedures*—Occurs frequently but will not be tabulated.
4. *Secondary to disease of lung, pleura or adjacent organs or tissues*—Six cases.
5. *Occurring in the apparently healthy*—Nine in the past three years; five others presented.

Though to-day's discussion deals rather with the two latter classes, the first and third are closely related and illustrative cases are presented.

CLASS 1. TRAUMATIC.

1. M.P. age 19. Admitted March 11, 1933. Driving a motor car which hit something throwing her against steering wheel. Pain in back. Became hoarse. Short of breath. X-ray 54110 almost complete pneumothorax on left side. No fluid. No fracture. Lung clear. Good recovery. No evidence of pulmonary disease.
2. J.D. Admitted December, 1932. Struck by truck, fracture of left 7.8.9 ribs. December 20, X-ray 52467 showed pneumothorax with slight fluid. December 31, X-ray 52466 showed fluid to 5th rib, lung compressed. January 12, X-ray 52878 showed slight fluid, air space $1\frac{1}{4}$ inches

at periphery. February 28, Fluoroscopic—fully expanded. No evidence of pulmonary disease.

3. G.W. Age 21. Admitted December 7, 1930. Motor accident. Increasing dyspnea—valvular opening. Aspiration—use of manometer to regulate intrapleural pressure. Good recovery. No evidence of pulmonary disease.
4. T.T. Age 18. Admitted August 22, 1930. Trauma left chest. Bullet. Fluid sterile. Accident August 7, slight fever to 10th. Sixty ounces bloody fluid aspirated August 17th. Good recovery. No evidence of pulmonary disease.
5. J.S. Age 43. Admitted December 3, died December 5, 1931. Thrown off wagon and dragged. Fracture 8.9.10 right ribs. Rupture liver. Right pneumothorax found at autopsy. No pulmonary disease. X-ray 45680.
6. J.C. Age 47. Admitted December 24, 1932, died December 26. Right pneumothorax. Surgical emphysema. Laceration forehead, fracture left mandible. Many abrasions and contusions. Fracture of 3.7 ribs on right side. Bronchopneumonia.

CLASS 2. INDUCED—(THERAPEUTIC). Not discussed.

CLASS 3. ACCIDENTAL IN COURSE OF THERAPEUTIC PROCEDURES.

I think these occur much more frequently than is recognized both in the course of (1) artificial pneumothorax and in (2) thoracentesis for the discovery or removal of fluid.

1. In a paper just published, Karan of the Rhode Island State Sanatorium reports (American Review of Tuberculosis October, 1933) that in a one year period seventy pneumos were induced. Spontaneous pneumothorax occurred in nine, or thirteen per cent. before the first refill. In three of the nine no air was insufflated. None were the worse for the spontaneous pneumothorax.
2. A case illustrative of pneumothorax occurring in course of aspiration of pleural fluid was that of J.M. age 27. Admitted May 10, 1933. Pregnancy, after labour pneumonia with a right-sided serous effusion. Paracentesis was followed by a small pneumothorax. X-ray 49828. All resolved in 50439 a few weeks later.

CLASS 4. SECONDARY TO DISEASE OF THE LUNG, PLEURA OR ADJACENT ORGANS OR TISSUES.

1. A.V. Age 42. Admitted December 1, 1931. Died February 22, 1932. Fibrosarcoma of right arm with secondaries in lung. Left spontaneous pneumothorax. X-ray 46602.

Tuberculosis of the lung is the most frequent disease causing spontaneous pneumothorax. We had five such cases in our wards since March last year.

1. A.A. Admitted April 6, 1932. X-ray 48043. Following his pneumothorax he developed fluid in the pleural cavity. Tubercle bacilli were present in both sputum and pleural fluid.
2. R.L. Age 29. Admitted May 13, 1932. Died June 13, 1932. Far advanced bilateral tuberculosis. One week before admission while eating breakfast had sharp stabbing pain in back right chest with dyspnea. No X-ray. Confirmed at autopsy.
3. A.Z. Admitted March 16, 1932. Sudden pain January 11th left chest. Developed pleural fluid. Tubercle bacilli present in fluid and in sputum. Bilateral pulmonary tuberculosis. Transferred to Sanatorium. X-ray 46514.
4. M.B. Out-patient. When first seen had partial pneumothorax *left base*. Absorbed. X-ray 54602. Sputum over thirty specimens negative. In this case the first X-ray films suggested a possible tuberculous etiology but a study of the films when lung was fully expanded resulted in the radiographic report of "insufficient on which to make a diagnosis of tuberculosis".
5. H.F. Admitted May 8. Died May 10, 1933. Cough and sputum eight months. Alcoholism eight years. Complete pneumothorax left side. All right lung involved in caseous disease. No X-ray.

CLASS 5. SPONTANEOUS PNEUMOTHORAX OCCURRING IN THE APPARENTLY HEALTHY. These following cases illustrate this group in which there is no demonstrable disease or etiological

agent, who promptly get well and appear to be good insurance risks except for the rare case such as Miss W. who had pneumothorax develop first in 1926 on one side then on the other side, and neither has absorbed completely to date, seven years later.

1. Mrs. J.M.O. Seen in consultation with Dr. W. E. Ogden. Right-sided pneumothorax February, 1920, when eight months pregnant. No evidence tuberculosis. X-ray on glass plates have disappeared but she has been followed closely by X-ray and all serological tests and never has shown any evidence of tuberculosis.
2. W.McE. Seen in consultation with Dr. J. A. Duck. Left pneumothorax June, 1920. When at breakfast pain in left side increasing as he went to street car to go to work, on reaching office had to be sent home with increasing dyspnea and cyanosis. Relieved by aspiration of air at intervals, until, I presume, the rupture healed. Examined at intervals since, no evidence of tuberculosis or other disease.
3. C.J.O. Seen in consultation with Dr. R. E. Joyce May, 1924. Sudden onset with pain lasting two days, gradually increasing dyspnea relieved by thoracentesis and allowing air to escape. Developed serous fluid which rose to 5th rib by June 16th. Fluid sterile. X-ray negative for disease 1924 and 1927. In perfect health to-day.
4. J.K. Age 43. Admitted August 11, 1925. No previous illness. Pain in left chest eight days before admission increased with walking. X-ray 19097 August 10th showed large left pneumothorax. No evidence of tuberculosis. Lung expanded promptly.
5. W.C. Age 25. Admitted December 26, 1926. Discharged January 11, 1927. Severe pain in right chest three weeks previously. Right pneumothorax. Right lung partly collapsed. X-ray 22497. Lung fully expanded. No evidence of disease.
6. G.P. Age 34. Admitted June 13, discharged June 21, 1927. While at work as labourer May 20 sharp pain upper

left chest, had to stop work, bed fifteen days. History of similar pain in Montreal three years before but in bed only one week then. Small amount of fluid. No evidence of disease. Not seen since.

7. M.L. Age 16. Admitted February 27, 1930, for tonsillectomy. A large right pneumothorax discovered. No symptoms of onset. X-ray 35568 March 1, no fluid. X-ray 36147 April 5, slight fluid, doubtful Tbc. After history negative.
8. S.B. Age 60. Seen with Dr. L. J. Breslin September 14, 1931. Pain seven weeks previously in shoulders and back of neck as of diaphragmatic pleurisy. Ten weeks previously ileus which followed operation for a small ventral hernia and an inguinal hernia. X-ray no evidence of disease. Air absorbed in three to four weeks. In perfect health to-day.
9. E.M.H. a physician seen in July, 1931. Gradual onset of pneumothorax in the course of his work. Went on to complete collapse of one lung. Gradual re-expansion. No evidence of clinical tuberculosis but the opposite lung showed one large calcified shadow as of an old healed primary infection in childhood. Prompt recovery. Good health since.
10. G.S. In hospital November 13 to January 6, 1932. Five days before admission was perfectly well, then a pain in the chest, a cough and a gone feeling. X-ray 45442 right basal pneumothorax, no definite tuberculosis. January 2, X-ray 46085 no evidence of disease. Cannot find anything to justify a diagnosis of tuberculosis.
11. A.MacK. Admitted April 21, 1932, after a drunken brawl, with fracture of mandible. On night of April 25 awakened in bed in hospital with a sharp pain in right chest, cough and dyspnea. A complete pneumothorax developed on the right side. His cough still present August 15th. No definite tuberculosis. Is still under observation at Chest Clinic. X-ray 48079 and 48381.

12. T.T. Admitted August 29, 1932. Pain in right chest ten days previously when at work, not anything strenuous. Short of breath on exertion. August 29 X-ray 50189 right pneumothorax. September 16 X-ray 50568 fully expanded. In good health June 26, 1933. No evidence of pulmonary disease.
13. L.S. Age 16. Seen in consultation with Dr. A. A. Cauley and Dr. J. A. Bauer, Hamilton, March, 1933. Onset of pain and dyspnea in gymnasium, gradual complete right-sided pneumothorax. Absorbed by April 20th. No evidence of tuberculosis in series of films repeated to date.
14. M.G. Seen September, 1933, in consultation with Dr. Chaikof. Sitting at table in hotel awaiting serving of his dinner taken with acute pain in chest and through to back, increasing in intensity with dyspnea and signs of shock. Went on to complete pneumothorax and collapse of lung. No evidence of pulmonary disease.

In presenting you concise case records with X-ray films of twenty cases of spontaneous pneumothorax seen in a little over three years, nine are shown which are classed as spontaneous pneumothorax occurring in the apparently healthy. Five others are presented with a longer history up to thirteen years, all of whom are in excellent health to-day.

DR. FELLOWS—From an insurance standpoint, I should say that if you have the history of an individual who has had a pneumothorax and if at the time there was no X-ray evidence of an underlying pulmonary pathology and no recurrence in perhaps a couple of years, the odds would be in favor of your dealing with a normal individual who had had an accidental medical incident for which we have no logical explanation and which probably will not interfere with his life history.

QUESTION—Did any of the cases show fluid?

DR. FELLOWS—Yes, they did.

DR. EADS—It might be interesting to mention four cases I saw last year. They were all young people in their twenties. Three were employees of the Penn Mutual, one was a medical student. One occurred when a young chap ran up a flight of steps; another was as the result of an injury; the other two were spontaneous for no apparent reason whatever. No fluid was found. One was a tuberculosis case. They all, with the exception of the tuberculosis case, recovered in a very short time and all returned to work.

DR. SCADDING—The next paper will be presented by Dr. John T. Eads who, in addition to being a valuable member of the medical staff of the Penn Mutual Life Insurance Company, is also Lecturer in Medicine at Jefferson Medical College and Clinical Assistant at both the Jefferson and Pennsylvania Hospitals.

THE SIGNIFICANCE OF EPIGASTRIC PAIN

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Abdominal pain is a very common complaint in the histories of patients hospitalized for diagnostic survey and treatment. Of these cases in which abdominal pain is common, pain more or less localized to the epigastrium seems to be found the greatest number of times. We have long considered pain in the abdomen as significant of some intra-abdominal organic lesion. This, of course, is true of those cases in which the symptoms are severe and in which the composite picture is one of definite pathological significance. There are those cases in which the symptoms are relatively mild and not persistent at the one extreme, and then there are those in which severity, persistency and recurrences are the chief characteristics at the other extreme. To make a survey of a number of cases in which epigastric distress is the chief feature of the history is the purpose of this paper.

In over 1,100 cases referred for gastro-intestinal studies in the Jefferson Hospital of Philadelphia 650 had as their chief complaint some form of abdominal pain. Many more than these 650 cases had abdominal discomfort associated with other symptoms, but only those cases in which abdominal pain was the chief complaint have been considered here. This article is based on a consideration of these 311 cases in which the history of the present illnesses began with pain in the epigastrium.

In these cases the length of the histories varied considerably. The longest duration of recurring symptoms was twenty-four years, the shortest three weeks. For the purpose of convenience this time element has been averaged for each group of lesions.

The accuracy and completeness of the histories in each of these cases has been of considerable interest.

There is a distinct difference in the percentage of error between clinical diagnoses and the confirmed diagnoses in these cases in which length and completeness of the histories and phy-

sical examinations have been sacrificed for lack of time and for pressure in the examination of too many patients. This is exemplified by a comparison of those histories of ward patients with the ones observed in the out-patient departments. The percentage of error in the diagnoses has been much greater in the latter.

Many of the cases earlier reviewed have been 'repeats'. Cases which have been studied several times over a period of several years without definite diagnoses being made at first. In these cases the persistence of the symptoms occurring over a long period with, of course, remission periods of comparative freedom from symptoms, have finally led to a finding of a definite pathological entity to account for the symptoms. It is these cases which are particularly interesting in their unquestioned proof of the importance of the symptoms complained of.

The importance in each case of obtaining a detailed history from each patient from the very incipency of their trouble to the time of their present diagnostic survey can not be over-emphasized. Many histories in the beginning seem insignificant, yet in the final analysis are quite important in summing up the final survey. There are cases in which the earliest symptoms were of such a mild nature that they are considered to be of no great importance. Cases in which some dietary indiscretion was considered to be the cause of the trouble. Yet, in how many such beginnings have serious organic lesions had their inception?

We are familiar with the fact that lesions anywhere in the abdomen can produce upper abdominal distress. The fact that lesions not intra-abdominal also can produce epigastric pain is also known. According to Morley the mechanism for abdominal pain is a dual one. He considers two types. The one type being poorly localized and indefinitely described. This type is the visceral type of pain. The sympathetic nervous system undoubtedly plays a part in this type of pain. Morley believes that visceral pain is most apt to be felt where the point of sensibility is the highest. He believes that visceral pain is of a relatively low sensibility and that by sympathetic radiation is carried to a point of higher sensibility where it is localized by the individual. This

point of higher sensibility is usually in the solar plexus area, hence the more or less localization of the pain to the epigastrium. No one can explain why in some instances there is radiation of pain and in other instances there is no radiation of pain. The whole question of referred pain is one which is by no means clear.

The other type of pain, according to Morley, is due to irritation of the parietal peritoneum. This type of pain is readily localized to the point of irritation.

The study of this series of cases of epigastric pain proves definitely that upper abdominal discomfort whether mild or severe should be investigated thoroughly. It will also show the difficulty in arriving at a satisfactory diagnosis in many instances. The multiplicity of lesions and the high percentages of organic lesions responsible for upper abdominal complaints are of interest. We have long known the syndrome of symptoms inseparably linked with well-known lesions of the upper digestive tract. It is of interest to observe in some instances where the symptoms were more or less typical of some lesion and yet were definitely proven to be due to something else which is not usually known to give such a syndrome.

We are all familiar with the lesions of the digestive tract which are the most common in giving upper abdominal distress. We are also aware of certain cases of pathology above the diaphragm which are more or less masked by abdominal symptomatology. Cabot in over 2,500 cases of abdominal pain reported almost 900 due to cardiac disease. Cabot in his case reports shows that cardiac disease, the majority of which were vascular, such as coronary artery disease, could and did produce symptoms which were primarily digestive or abdominal in their symptomatology. In a recent review of over 400 cases of heart disease by Hamilton in a recent paper before the Pennsylvania State Medical Society showed that 150 gave as their chief complaint upper abdominal distress. This has been found to be true by many other investigators.

By far the most common cause of epigastric pain was found to be some lesion of the digestive tract as would be expected.

Naturally, there are many other lesions than those mentioned in this series which are capable of producing upper abdominal symptoms. It must be understood that all of these cases are those in which the symptoms were such that they were considered to be, as above mentioned, due to some gastric-intestinal problem. Those cases in which the findings were such as to rule out a gastric-intestinal lesion were not seen and, naturally, not included in this series, so the value of this group lies in the final diagnoses of cases primarily considered digestive. Of interest is the difficulty in differentiating from history and physical findings alone the various digestive lesions responsible for the symptoms.

TABLE I

| CONFIRMED DIAGNOSES | CLINICAL DIAGNOSES | | | | | | |
|------------------------|--------------------|-----------------|------------------|--------|--------|---------|---------|
| | No. Cases | Peptic Ulcer | G. B. Disease | Cancer | Ap'dix | Colitis | Cardiac |
| Biliary Disease.. | 84 | 40 | 30 | 4 | 4 | 0 | 6 |
| Duodenal Ulcers | 60 | 28 | 20 | 8 | 2 | 2 | 0 |
| Gastric Ulcers.... | 15 | 4 | 5 | 2 | 0 | 2 | 2 |
| Cardiac Lesions.. | 47 | 10 | 12 | 5 | 0 | 0 | 20 |
| Malignancy | 40 | 10 | 5 | 19 | 0 | 0 | 6 |
| Appendicitis..... | 20 | 5 | 8 | 0 | 6 | 0 | 1 |
| Colitis | 10 | 3 | 0 | 0 | 3 | 4 | 0 |

In the above table will be seen the various lesions in this series of cases which cause the symptoms of upper abdominal distress. As will be seen, gall-bladder disease is the most common cause according to this study of epigastric pain. This was found to be true in 84 cases. Of these, 34 were men and 50 were women. Sixty of these were overweights. Thirty-three of these cases were complicated by calculi. Sixteen of these patients complained of weight loss. The average length of the symptoms ranged around five years. Thirty-four cases had a low gastric acidity; 16 cases had a normal acidity and 14 cases a hyperacidity. The provisional diagnoses in these 84 cases of proven gall-bladder

disease were as follows: Forty were diagnosed peptic ulcer, 4 cancer, 4 appendicitis, 6 as cardiac lesions and 30 were diagnosed as gall-bladder pathology. These diagnoses were based on history and physical findings. Of the 40 cases diagnosed clinically as peptic ulcers, 36 were considered to be duodenal ulcers. Of these cases diagnosed as duodenal ulcer the history of the symptoms ranged from five to seven years. In these cases the relationship of pain to the taking of food was that commonly thought as being that of the peptic ulcer syndrome. Night pain occurred in 16 cases and in these cases diagnosed as duodenal ulcer occurred all of the 14 cases of hyperacidity. This fact to some extent possibly can explain the ulcer-like syndrome of symptoms. The differential diagnoses were made only after special studies and at the operating table.

Thirty of the 84 cases definitely proven to be gall-bladder pathology were so diagnosed clinically.

The fact that many gall-bladder cases are diagnosed as cardiac cases is exemplified in six cases in which the symptoms were diagnosed as coronary artery disease. No evidence was found to support this diagnosis and definite gall-bladder pathology was demonstrated.

The age limits of these cases of gall-bladder disease ranged from 24 to 62 years with the greatest number being between forty and fifty years.

Ordinarily, it has not been considered a very difficult feat to differentiate between gall-bladder disease and peptic ulcer. However, in these 36 cases diagnosed clinically as duodenal ulcer the history and physical findings were certainly more nearly the typical ulcer syndrome than some of the ulcer cases themselves,—pain coming on two or three hours after meals relieved by food; pain at night awakening the individual, loss of weight; an average history of five to seven years duration; a hyperacidity; no right sided pain or shoulder tip pain, jaundice or any other common gall-bladder phenomenon. These cases are only a few but they demonstrate how difficult it is to make an accurate clinical diagnosis without special studies.

In these cases of definite gall-bladder disease there were 18 which can be classified as 'repeats'. Of these 18 cases 8 were cases of calculi. These 18 cases were studied at different times thoroughly over a period of five years before definite conclusions were reached. All the time their symptoms persisted with very little relief. In the cases of calculi from two to four different X-ray studies were done before the stones were found. These 18 cases had persistent, recurring symptoms over a period varying from twelve years to five years in duration. Yet it was only after a five-year observation that organic pathology could be definitely proven in some instances.

The diagnosis of appendicitis was made in 4 cases of gall-bladder disease. Three of these were operated and while there was probably some appendiceal involvement yet most of the pathology was in the biliary tract and in these cases the chief complaint was epigastric distress.

Appendicitis is quite capable of causing discomfort in the epigastrium. We are all familiar with the early pain of acute appendicitis which is apt to be in the epigastrium. Of course, this as a rule becomes localized to the right lower quadrant. However, in a number of cases of chronic appendicitis which were included in this series 5 were diagnosed as peptic ulcer and 8 were diagnosed as gall-bladder disease. In these cases the history was about three or three and one-half years duration. They complained of epigastric discomfort. Their symptoms were all those of upper abdominal lesions. These cases at operation showed only appendiceal pathology.

Of this series of 311 cases of epigastric distress supposedly of digestive origin 47 were found to be due to some cardiac lesion. Twenty of these 47 cases were diagnosed clinically as probable cardiac cases but were referred for gastro-intestinal series to rule out possible gastro-intestinal pathology. The symptoms in the remaining cases were so masked that a cardiac basis for the complaint was not considered. This is a relatively low number of cases as compared to those found by other investigators, but it must be remembered that this clinic was primarily a gastro-intestinal clinic and not a cardiac clinic.

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The age limits of those cardiac cases ranged from 38 to 67 years. As would be expected the largest number fell in the period from 45 to 55 years.

The symptoms of upper abdominal distress frequently accompanied by flatulent indigestion, often with definite relation to meals and sometimes associated with definite weight loss, often led to a clinical diagnosis of peptic ulcer, malignancy or gall-bladder disease. The age of the individual and the general picture of the case together with the misleading symptomatology made a clinical diagnosis difficult.

Of interest from a cardiac standpoint were three cases in which the clinical diagnoses were vague. These consisted of a picture of possible faulty dietary and living habits or of gastric neuroses. The studies from a gastro-intestinal point of view were entirely negative. Two of these cases came to post-mortem with the post-mortem diagnosis of coronary artery disease. Their ages were 43 and 47, respectively. This merely exemplifies the caution with which any upper abdominal symptoms in individuals past the age of forty should be regarded.

There were 9 cases in the 27 cases of cardiac lesions not clinically diagnosed, which had several studies made over a period of three years. All these studies were gastro-intestinal and all were negative. These were done before definite cardiac pathology was proven. Their symptoms meanwhile were persistent. In such cases only repeated studies were of value in reaching any definite conclusions.

Cabot in his 2,500 cases of epigastric pain found that pain due to a heart lesion was so prevalent that the term 'angina abdominalis' was applied to such attacks.

In these cases of cardiac lesions in which no suspicion of cardiac involvement was apparent clinically the only means of making an accurate diagnosis was by making special studies such as an electrocardiographic study, X-ray examinations, etc.

Cases in which cardiac damage was suspected were those in which a definite valvular lesion could be demonstrated. The average history of trouble ranged around eighteen months to two years. However, in most instances the histories went back fur-

ther with definite symptoms of digestive upsets, flatulence, etc., for some time leading to a suspicion of gastro-intestinal trouble.

The 27 which were not diagnosed clinically as heart cases had the final diagnoses made as follows: coronary artery disease 15 cases, mitral stenosis 4 cases, aortic disease 3 cases, 4 cases of myocardial degeneration with liver changes, 1 case of pericarditis. It is of interest to note that 20 of these 27 cases were overweight individuals.

There were 75 cases in this series of 311 cases comprising those definitely diagnosed as peptic ulcer cases. Sixty of these were duodenal ulcers and 15 were gastric ulcers. Of these 60 duodenal ulcers 45 were in men and 15 in women. Of the 15 cases of gastric ulcers 11 were women and 4 were men. The age limits in this series of peptic ulcers were 22 to 54 years with the majority occurring in the decade from 30 to 40 years. Of these 75 cases of peptic ulcers 68 of them were underweights, 55 were of the asthenic type of build. Loss of weight was a chief symptom occurring in 70 cases.

The average history of symptoms in the duodenal ulcer cases was seven years. The average history of symptoms in the gastric ulcer cases was five years.

In the duodenal ulcer cases 50 had hyperacidity, 44 complained of night pain, 46 had pain definitely related to meals occurring within the time limit of the usual duodenal syndrome.

In the gastric ulcer cases 9 had hyperacidity, 8 complained of night pain, 11 had pain definitely related to meals of the type seen in many gall-bladder cases.

In these cases of duodenal ulcers the provisional diagnoses were as follows: gall-bladder disease 20 cases, duodenal ulcer 20 cases, gastric ulcer 8 cases, cancer of the stomach 8 cases, colitis 2 cases, appendicitis 2 cases.

In the clinical diagnoses of 15 proven cases of gastric ulcer were gall-bladder disease 5, gastric ulcer 2 cases, duodenal ulcer 2 cases, cancer of the stomach 2 cases, coronary artery disease 2 cases, colitis 2 cases. It is interesting to note the relationship between gall-bladder disease and duodenal ulcer from a clinical diagnostic point of view. In the 84 cases of proven gall-bladder disease 36 were diagnosed duodenal ulcer. In turn, in the 60

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cases of proven duodenal ulcer 20 had been previously diagnosed as gall-bladder disease.

Only 20 of the 60 cases of duodenal ulcer were so diagnosed clinically. Of the 15 cases of gastric ulcers only 2 cases were diagnosed clinically. Here again gall-bladder disease was the most frequent diagnosis made.

The difficulty in distinguishing between peptic ulcer and gall-bladder pathology is apparent.

It is interesting to note that 6 of the 15 gastric ulcers occurred on the greater curvature side of the stomach; all were between the ages of 30 and 40 years and all were proven to be malignant. This is a finding which of late has been considered as being almost always true of greater curvature ulcers.

There were 11 cases of proven duodenal ulcer which had been studied before. The average length of duration of their symptoms was eight years. Over the period from 1928 to the present there had been from two to four complete gastro-intestinal studies on each of these cases with no definite diagnoses made. The diagnoses of hyperchlorhydria, dyspepsia, gastric neuroses, etc., had been made over this period. There was nothing definite enough found to warrant an absolute diagnosis being made. Surgery was not considered. These cases were finally diagnosed as duodenal ulcers. This merely shows that repeated efforts must be made to find the lesion responsible.

There were 40 cases in the series of 311 cases which were malignant. Of these 34 were gastric, 2 pancreatic, 1 gall ducts, 1 liver, 1 esophagus and 1 retro-peritoneal sarcoma involving the stomach and duodenum.

The age limits were 27 years to 68 years. It is interesting to note that there were 3 cases of gastric carcinoma between 27 and 30 years of age. Of course, the greatest number of cases occurred between the ages of 45 and 55 years. There were 26 men and 14 women. Loss of weight was the predominating symptom.

It is of interest to note probably one of the most frequent conditions which causes confusion in diagnosing these malignant conditions, aside from peptic ulcer and gall-bladder disease, was a clinical diagnosis of a cardiac lesion. Naturally, the age of the patient and general picture of the case were confusing.

The clinical diagnosis of these 40 cases of cancer involving the gastro-intestinal tract were peptic ulcer 10 cases, gall-bladder disease 5 cases, cardiac disease 6 cases and the remaining 19 cases were clinically diagnosed as cancer.

In this series of 311 cases there were 10 cases of proven colitis which gave upper abdominal symptoms. The fact that colitis often gives epigastric distress and upper digestive symptoms leads to frequent errors in diagnosing.

An irritable colon is capable of giving a picture which from an X-ray point of view is almost identical with the deformity of the duodenal cap seen in an ulcer.

The most frequent clinical diagnosis made on these 10 proven cases of colitis were peptic ulcers in 3 cases, appendicitis in 3 cases and colitis in 4 cases. Four cases were operated upon and no pathology found anywhere in the upper digestive tract or in the appendix.

There was a group of 20 cases in the series which might be grouped under miscellaneous cases. They were as follows: tuberculosis of the cecum 1 case, partial intestinal obstruction 2 cases, cirrhosis of the liver 2 cases, viscera optosis 3 cases, duodenal stasis 2 cases, aneurism of the aorta 2 cases, hyperthyroidism 2 cases, carcinoma of the lung 1 case, post-operative adhesions 2 cases, renal calculi 3 cases.

All of these cases complained of upper abdominal distress or other digestive symptoms. They were all primarily considered as lesions of the upper gastro-intestinal tract. Gall-bladder disease, peptic ulcer and appendicitis being the chief clinical diagnoses in these cases.

In this group of cases can be seen the variety of complaints capable of giving epigastric distress.

There was a group of 15 cases in which a diagnosis could not be made. These cases were diagnosed as follows: peptic ulcer 5 cases, gall-bladder disease 6 cases, appendicitis 1 case, colitis 1 case, viscera optosis 2 cases. All the studies in each of these cases were entirely negative. Two cases were operated on and nothing found. There were nine women and six men. The age range was from 17 years to 44 years.

TABLE II

| LESIONS | Men | Women | BODY BUILD | | Pain and Meals | Night Pain | Average Duration Symptoms | Gastric Acidity | | Shoul- der Pain | Loss of Weight |
|-----------------------|-----|-------|---------------|--------------|----------------------|---------------|---------------------------------|--------------------|----|-----------------------|----------------------|
| | | | - Asthenic | + Sthenic | | | | + | - | | |
| Biliary Disease | 34 | 50 | 0 | 60 | 40 | 16 | 5 yrs. | 14 | 34 | 64 | 16 |
| Duodenal Ulcer | 45 | 15 | 45 | — | 46 | 44 | 7 yrs. | 50 | — | 6 | 57 |
| Gastric Ulcer | 4 | 11 | 10 | — | 11 | 8 | 5 yrs. | 9 | — | 1 | 13 |
| Cardiac | 32 | 15 | 4 | 30 | 30 | 4 | 2 yrs. | 5 | 6 | 3 | 22 |
| Malignancy | 26 | 14 | 29 | 6 | 37 | 10 | 1½ yrs. | 2 | 33 | 3 | 38 |
| Appendicitis | 12 | 8 | 8 | 8 | 5 | 0 | 2½ yrs. | 4 | 1 | 1 | 8 |
| Colitis | 7 | 3 | 6 | 2 | 3 | 1 | 3 yrs. | 2 | 1 | 0 | 5 |

TABLE III

| LESIONS | No. of Cases | AGE GROUPS | | | | | X-rays and Studies Positive | Number of Cases Operated | Post Mortem |
|-----------------------|--------------|------------|-------|-------|-------|-------|-----------------------------|--------------------------|-------------|
| | | 20-30 | 30-40 | 40-50 | 50-60 | 60-70 | | | |
| Biliary Disease | 84 | 5 | 25 | 44 | 9 | 2 | 78 | 48 | 4 |
| Duodenal Ulcer | 60 | 10 | 29 | 14 | 6 | 1 | 54 | 42 | 3 |
| Gastric Ulcer | 15 | 4 | 7 | 3 | 1 | 0 | 15 | 12 | 3 |
| Cardiac Lesions | 47 | 0 | 2 | 18 | 25 | 4 | 47 | 2 | 11 |
| Malignancy | 40 | 3 | 6 | 11 | 15 | 5 | 38 | 28 | 15 |
| Appendicitis | 20 | 9 | 8 | 3 | 0 | 0 | 12 | 17 | 0 |
| Colitis | 10 | 4 | 4 | 1 | 1 | 0 | 9 | 2 | 1 |

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In Tables II and III the various lesions in the series of 311 cases of epigastric pain have been grouped according to various points of interest, i.e., history, physical findings, age, duration of symptoms, et cetera. From these cases can be seen in some cases the variation in some respects from our text book pictures and in other cases the accepted syndrome of the lesion as taught us.

From the above discussion it will be seen that 296 of the 311 cases studied were proven to have some organic lesion responsible for the symptoms. Only 15 of the 311 cases were entirely negative in every respect. It must be realized that all of these patients were sick and that they were either clinic or ward patients, hospitalized for diagnosis and treatment. In almost every case the symptoms were of a fairly long duration, and in almost every case there was unquestionably a severity of symptoms. Of interest are the number of cases which in their very beginning were considered of no serious significance. Practically all of these cases have been under more or less careful medical attention for years for digestive trouble. Most of them had been told at one time or another that there was nothing seriously wrong. Very few of these cases with the exception of the few 'repeats' seen in the hospital over the past five years had been studied carefully. Many of these cases had 'passed' examinations for insurance. Yet with the exception of a few patients all were definitely proven to have an organic lesion present.

Unquestionably, some dietary indiscretion, some improper elimination or some focus of infection can temporarily cause abdominal distress. It is the persistence of these symptoms and their tendency toward recurrence that should be cause for extreme care in passing on the presence or absence of organic pathology. It is such cases that call for careful inquiry into every detail of their history, for an accurate and thorough physical examination, and for any additional special studies. Every case of organic disease capable of causing epigastric distress has at one time or another been rather mild and insignificant. They very seldom begin as 'full-bloom' cases of such definite proportions that they can be adjudged organic at first glance, but year by year, month by month, the symptoms persist. They may be temporarily re-

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lieved but they return. With such cases it is dangerous to temporize.

CONCLUSIONS

1. Epigastric pain in sick individuals is significant of organic pathology, and in supposedly healthy individuals should be a cause for suspicion if there are repeated attacks.
2. The most common cause of epigastric pain is a lesion of the gastro-intestinal tract.
3. The most common cause in this series of cases is gall-bladder pathology.
4. The second most common cause is peptic ulcer.
5. The fact that extra-abdominal lesions can cause epigastric distress is proven.
6. The variety of lesions capable of giving epigastric pain has been demonstrated.
7. The difficulty in differentiating between various lesions from history and physical findings has been shown.
8. The importance of a careful, complete and accurate history together with a thorough physical examination cannot be over-emphasized.

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DR. FELLOWS—Apparently a sure way not to have epigastric pain is to be an applicant for life insurance, if one believes all he hears.

In reviewing 1,688 first and second year death claims in which the death certificate stated that death was due to one or more of the many conditions which usually cause epigastric pain, not once did the admission of that symptom appear in the application papers. Furthermore, 100 other individuals who did admit some stomach disease, dyspepsia or indigestion when applying for insurance did not use the words epigastric pain and only rarely the word pain, nor did the examining physician use that term.

Among these first and second year claims, there were 1,434 without insurance record, abnormal history of physical examination and 254 with insurance record, unusual history or abnormal physical examination. Sixty-three of the death claims from the group which were entirely clean at the time of application were rejected after investigation brought out the true facts. Among the 254 cases which did have some insurance record, history, or physical abnormality, 81 cases were fully investigated and it was found the medical treatment or symptoms were present when the application was made for insurance. In the entire group of 144 investigated death claims, abdominal pain or other symptoms had been present prior to applying for insurance, but were not mentioned in the application papers.

Of course, we all know that many people have epigastric pain and many other symptoms or abnormal conditions, but when applying for life insurance they don't admit it. About the only point brought out in this discussion is the old one, that we must keep everlastingly at the improvement in medical history taking and the examination of the applicant.

DR. SCHOLZ—Dr. Eads' material was taken from a study of one of the largest hospital out-patient departments of this Continent. The total number of patients seen for the first time in that particular dispensary, for the year 1932, was 15,170. The total number of patients seen in the same period in the gastro-intestinal clinic was 4,860. While these percentages are not applicable to the ordinary or the average out-patient department they are significant of the morbidity of gastro-intestinal impairments, and suggest perhaps, that we are not obtaining from a

goodly percentage of our applicants, any reference to these conditions, which may, on the initial symptoms, seem trivial and unimportant, but which do, in many instances, after a period of years, progress to early mortality.

Dr. Eads has spent months in collecting material which was used for the development of this manuscript and I believe his study of "The Significance of Epigastric Pain" is of particular interest to us. He was careful to exclude from his data all patients except those individuals with an entry of epigastric pain. With the exception of Dr. Cabot's work I have not found in the literature a comprehensive study of a large group of patients whose only symptom on consulting a physician was epigastric pain, a reference to which occasionally occurs in our examination reports.

Each of Dr. Eads' conclusions are well worth careful thought, but to me the most important thing for us to remember in connection with his paper is the last notation under his conclusions—"The importance of a careful, complete and accurate history, together with a thorough physical examination cannot be over-emphasized".

Regardless of the development in roentgenological and laboratory methods in determining accurate diagnoses of gastro-intestinal conditions, and regardless of how carefully the studies are interpreted, we are still faced with the importance of personal histories. In life insurance particularly do we receive too short, incomplete histories. We do not, or rarely, have well-defined, consecutive statements of symptomatology during the years the applicant has suffered intermittent epigastric or upper abdominal pain. The histories would be of much value, and the time will soon come when we will all require them.

Many years ago I was greatly impressed by the number of applicants who had bilious attacks, and when, while still a novice, I spent considerable time in the archives of one of our larger companies. Here I unearthed surprising information as to what serious abdominal conditions were described as "bilious attacks". Later on there seemed to be a scheme to diagnose epigastric pain, whether or not accompanied by other symptoms, as "ptomaines"; yet on

inquiry we would frequently learn that no other member of the family partaking of the same food had ptomaine poisoning.

We have all had the experience of time and time again reviewing death claims with gastro-intestinal histories not completely developed, or to which proper significance was not given by the attending or examining physician, resulting in an unexpected, untimely, gastro-intestinal death claim. If we could impress our examiners with the necessity of well-taken, carefully noted personal histories, or if there were some way in which we could always obtain them, we would experience a mortality savings.

With mortality trends constantly changing and pursuant, shall I say, to a slightly changed selection procedure, the Medical Department of my Company has been more extendedly and more carefully developing personal histories. This is accomplished by further correspondence with the examiner for the development of personal histories, by emphasizing the necessity of the pertinent details of each and all attacks, and by obtaining from attending physicians, as far as possible, details of symptoms, diagnosis, treatment, number of attacks, etc.

Studying the declined ratio for one year—April, 1932, to March, 1933, inclusive—8.8 per cent. of all our medically declined applicants were declined because of gastro-intestinal histories. The medically rated percentage of substandard issues because of gastro-intestinal histories was 5.3 per cent. of the company's total medically rated issues. Of the combined groups, the declined and substandard issues, 36 per cent. of the cases would have been accepted standard had we been content to accept, without further data, the histories originally submitted by the examining physicians. This clearly indicates to us the advisability and necessity of completing the history by correlating the efforts of our examiners in developing careful histories with the information obtained from attending physicians.

When one considers that various authorities have reported that the symptoms of gastro-intestinal pathology are first noted from five to nine years previous to a diagnosis of a pathological gastro-intestinal impairment, the significance of epigastric pain in connection with disability claims is obvious. A study, as of July 17,

1933, of our policyholders incapacitated by gastro-intestinal diseases—to whom we are paying disability benefits—shows that this group is six per cent. of the total to whom we are paying disabilities. This includes only those disabilities qualifying as purely gastro-intestinal ones, all the combined impairments into which gastro-intestinal diseases entered being excluded. A crude, but as close a classification as could be made, indicated the following:

| | |
|-------------------------------------|-----|
| All ulcers (gastro-intestinal)..... | 38% |
| Colitis | 22% |
| Gall bladder infections..... | 18% |
| Carcinoma | 8% |
| Miscellaneous | 14% |

The miscellaneous group is composed of individuals who are incapacitated, and in whose cases there has been a difference in medical judgment as to the exact gastro-intestinal impairment. A few of these are combined gastro-intestinal impairments.

Another surprising and perhaps interesting thing, is that eighty-six per cent. of these policyholders are Anglo-Saxon; eight per cent. Semitic; four per cent. Slavic; and two per cent. Latin. Eighteen per cent. of these policyholders receiving disability benefits live in cities of from ten to twenty-five thousand population, while twenty-three per cent. are residents of small towns or country districts. Twenty-five per cent. of these individuals are residents of cities of twenty-five thousand or more population.

A study of the company's death claims for the three years, 1930-31 and 32, covers 9,725 lives. Forty-two per cent. of these policyholders died of cancer of the stomach, liver, intestines or pancreas; and in reviewing these death claims we felt that in a goodly number of the cases had the examiner and the home office worked up the history the individuals would not have been accepted as risks. Of the other gastro-intestinal diseases there were quite a number giving significant histories referring to the gastro-intestinal system, undoubtedly quite a number whose only early symptom was epigastric pain. Again, these histories were not, perhaps, as well developed as they could have been. This

group comprised six per cent. of the total number of death claims during these years.

In Dr. Eads' study of 311 cases of original entry epigastric pain, all of which were presumed to be of digestive origin, forty-seven of the individuals were found to have cardiac lesions. A careful study of these cases eliminated any gastro-intestinal pathology. This is fifteen per cent. of the cases in Dr. Eads' study, which is very high I believe, as compared to our group of applicants for life insurance.

Authorities vary in their judgment as to the percentage of cardiac conditions masquerading gastro-intestinal-wise. Their statements vary from two to five per cent. Studying our company's death claims for the three year period (a total of 9,725 lives) we find that approximately only one per cent. of the acute epigastric pain deaths terminated as cardiacs.

In concluding my few remarks in reference to the timely and worthwhile paper of Dr. Eads may I take the liberty of again inviting your attention to the duration of gastro-intestinal symptomatology in connection with gastro-intestinal pathology, the morbidity and mortality entailed, and the necessity, in this group especially, of the importance of careful, complete and accurate histories, as well as thorough physical examinations.

DR. EADS (closing)—In spite of the fact that Dr. Fellows found a large number of cases in which there was no complaint of abdominal pains, yet, clinically (I am not speaking from an insurance point of view when I say this), abdominal pain is a common complaint and as I said, of those 1,100 cases I reviewed, 800 had abdominal pain as a complaint. Whether or not it is due to the difference in the taking of the history, I am not able to say. Perhaps the clinician takes a different type of history from the insurance examiner. Perhaps the clinician goes into it and instead of taking bilious attacks as a complaint, goes in and develops this complaint of bilious attacks and, perhaps, working that way he develops the fact that so-called bilious attack was, primarily, pain or discomfort and perhaps that can account for the discrepancy between the cases on file in the insurance office and

these cases that were seen in the hospital. Then, again, the cases seen in the hospitals are cases of sick individuals—individuals who probably would not apply for insurance at that time. A great many of them had insurance. A great many of them had been passed and their history even went back prior to the time they secured the insurance. In seeing cases in the hospital clinics and in the outpatient departments, it is surprising the number of individuals who complain of some digestive trouble. In the outpatient department of the Jefferson Hospital last year, 15,000 cases were seen in the Medical Department. Approximately 6,000 were seen in the gastrointestinal department. Those were straight gastrointestinal cases. Of the 15,000 medical cases, a good number had digestive pains also on their complaint and I know from personal observation and personal experience that abdominal pain is a fairly common complaint.

In closing, I wish to express my appreciation of the privilege of being here and taking part in the discussion. I thank you.

SECOND DAY

DR. SCADDING—The next contribution to the program will be presented by Dr. Hunter, who, as you all know, is Vice President and Chief Actuary of the New York Life. He hails from bonnie Scotland, a country that has bred most of the world's greatest actuaries and indeed many of the world's intelligentsia.

He is, like most actuaries, bi-lingual at least. In spite of the fact that he has a greater medical vocabulary than I possess, when he speaks my language I understand him pretty well, but when he wanders off into Gaelic or Esperanto (or whatever the tongue peculiar to actuaries is called) I am completely lost! Most masters of science can, if they like you, make themselves really quite intelligible and most entertaining. This master happens to be a very good friend of all of us. I have a sneaking suspicion that he likes us. It is certain that we love him. He is an honorary fellow of this Association, and he assures me he will address us in plain English. The plainer, the better, concerning a subject so vital, in my opinion, to all of us whose chief job is selection.

COMMENTS ON
"MEDICAL IMPAIRMENT RATINGS"

By DR. ARTHUR HUNTER,
New York Life Insurance Company

My old friend Dr. Scadding has persuaded me to present a paper to you at this time although I had decided not to do so until new material had been developed. He has asked me to give you my views on the schedules of Medical Impairment Ratings published by the Joint Committee of the two societies. The title of this paper was chosen by Dr. Scadding and accepted by me without realizing the difficulty of making comments within reasonable limits on such a subject. To cover the fifty pages in the Medical Impairment Ratings and to refer of necessity to many of the eighty-one pages of text in the Medical Impairment Study gives one a rather hopeless feeling. After cutting down substantially the original draft I find myself with a sketchy paper which I hope, however, has covered some of the important matters to which attention should be called.

My first plan was to select certain impairments where it seemed that the work of the Committee required amplification, that additions might be made to the ratings, or that a different point of view should be expressed. On reflection, however, it seemed to be of much greater value if I were to collect the opinions of many minds, which would thereby reflect the general viewpoint on the selection of risks, as outlined in the M.I.R. It seems desirable in the first place to determine the measure of the success of the M.I.R. by ascertaining the extent to which it has influenced opinion and is of practical use. The result of the Committee's efforts might be determined by learning the number of companies which have adopted in whole or in part the suggested ratings. This does not limit the scope of the inquiry to those companies which employ the Numerical Rating System as the ratings of the Committee were intended to express their views of the relative mortality irrespective of each com-

pany's method of medical selection. I have accordingly asked the fifty largest companies which received copies of the M.I.R. the following questions:

1. Do you use the Schedule Ratings to the extent of 85% or more of the questions which arise in daily practice?

2. If not, do you use the book to the extent of 50%, but less than 85%?

3. If the suggested ratings are not incorporated into the Company's practice do you frequently refer to the Schedules for information or guidance?

4. Do you not use the publication at all or do you refer to it only very infrequently for information?

Another question was asked with which I shall deal later.

A synopsis of the replies is now given:

| | Number of Companies | Companies doing both standard and substandard business | Companies restricting their business to standard plans |
|-----------------------------------|---------------------------|--|--|
| 1. Use M.I.R. 85% or more..... | 26 | 24 | 2 |
| 2. Use M.I.R. 50% to 84%..... | 13 | 10 | 3 |
| 3. Refer frequently to M.I.R..... | 6 | 3 | 3 |
| 4. Seldom consult M.I.R..... | 5 | 0 | 5 |
| | — | — | — |
| | 50 | 37 | 13 |

Of the thirty-seven companies which issue policies to both standard and substandard lives, all but three use the M.I.R. to the extent of 50% or more and the other three use it frequently. Among the thirteen companies which limit their business to standard plans alone it is not a cause for wonder that five of them seldom have occasion to consult the M.I.R. On the whole it seems, judging from the number of companies which use the M.I.R. extensively, that it was worth the time and effort of the Committee, especially as it appears that these Schedules are in general use throughout the United States and Canada.

The first rating schedule prepared by Dr. O. H. Rogers and myself was completed 27 years ago. There have been many changes since that time in the treatment of individual impairments but, on the whole, our Company's experience on its under-

average lives has been close to that anticipated. Modifications have been necessary for various reasons, such as radical changes in medical or surgical treatment and the results thereof, greater recognition of the danger of certain diseases and more accurate information from our examiners. There might be mentioned as outstanding examples of the first of these, tuberculosis, the death rate from which has substantially decreased in the past quarter of a century, although the effects of malnutrition in these times may result in a temporary rise. There have been changes also in the degree of rigidity with which the ratings have been applied in our Company, but since the inception of the rating system judgment has been applied to cases which departed substantially from the average, and to impairments on which the data were scanty. In any schedules of this nature many of the modifications reflect the changes in medical and surgical opinion. Accordingly the physicians on our Joint Committee did not consider it advisable to incorporate the newest opinions in their ratings if there were no proof that they were correct.

In considering the differences of opinion with regard to the ratings published by the Joint Committee, it should not be forgotten that these are the composite opinions of fifteen members, Actuaries and Medical Directors. They represent a middle point of view based on adequate mortality investigation, clinical observation, limited statistics, or impressions gained by daily contact with the work, depending upon the importance of the subject and the available material. Each rating should, therefore, be considered not as an unvarying standard but as the combined wisdom, if we may so call it, of a group of the most experienced men in both professions.

One of the principal functions of the Committee was to bring more uniformity into the practice of the companies so as to avoid those aspects of competition which would be a detriment to both policyholders and companies. The interest of any company is not served by the common knowledge that its treatment is very liberal on a certain type of impairment as thereby the agents of many companies tend to send such business to it. Several companies have learned from an unfavorable experience that too much

liberality is costly under an impairment on which there was a wide difference of opinion. Every company should be free to make any experiments which it deems desirable, as in that way progress is achieved. The competition, however, which forces companies to grant terms too liberal in the judgment of the officers, is not only bad for the individual company but is apt to lead to a weakening in the morale of all those connected with the selection of risks.

IN WHAT CLASSES ARE THE MEDICAL IMPAIRMENT RATINGS TOO SEVERE OR TOO LIBERAL?

In order to determine whether, in the opinion of the Medical Directors and Actuaries of the companies, the ratings of the Joint Committee were considered too severe or too liberal, a question to that effect was asked in my circular letter. It was as follows: "In which of the principal impairments do you think the Committee was too liberal or too severe?" There were many expressions of opinion in the replies from fifty of the leading companies that the Committee's ratings were generally very fair. It is hardly necessary to mention particular impairments where a criticism was made by only one of the fifty companies. The following appears to be worthy of comment:

There were five companies whose officers thought the general ratings for heart involvements, including hypertrophy, were too severe and an equal number which thought them too liberal.

In the case of high blood pressure, fifteen companies suggested that the Committee's ratings were too liberal as against three which took the opposite position. Of the former, five referred specifically to diastolic pressures greater than 100 mm.

The opinions were equally divided in the case of pulmonary tuberculosis, four votes, and of the three classes of pleurisy, six votes.

Three companies suggested that the ratings were too liberal under cancer, leukoplakia and papilloma; two companies under epilepsy and insanity, and three under neurasthenia.

There were two companies whose officers were of the opinion that the Committee was too lenient under the general heading of

biliary colic and three that it was too severe under renal colic, if an operation had been performed.

The only other comments of general interest concerned duodenal ulcers where three companies considered that the Committee's ratings were too severe, and otorrhea where two thought them over-liberal.

It is rather interesting to note that the only medical director who expressed the opinion that the ratings for malarial fever should be reduced was with a company which had had the most extensive experience in malarial districts. The same was true of another medical director with regard to casts—his company has probably gone into that subject more thoroughly than any other.

Upon the whole there were 75 suggestions made, of which 48 (64%) were in favor of increasing the ratings here and there beyond those in the M.I.R. The other 27 (36%) favored a decrease in the ratings specified. Two-fifths of all the criticisms dealt with one or another of the circulatory impairments.

These comments give a bird's-eye view of the suggestions which have been received. Others mentioned particular points, which cannot be readily considered in a general survey.

A few of the comments in reply to the question under discussion (Are the ratings too severe or too liberal?) indicate that the function of a rating schedule is not entirely understood or that the statements made by the Committee in the introduction to the M.I.R. had been hastily read. For example, one of the objections raised was that the ratings were not based on experience. Reference was made to this by the Committee. The combined experience of all the companies in the country does not supply enough material for an assured rating on a number of types which occur infrequently. Another comment was to the effect that each applicant should be treated individually, but that does not bar the use of the ratings as a guide in practice. Still another comment was to the effect that as certain companies are taking slightly substandard cases at the standard rate of premium, contrary to the recommendations of the Committee, why should all not do so? The implication is that such companies, which are decidedly in the minority, are correct and that

the majority are wrong. As a matter of fact, these differences occur in comparatively few types, such as overweight, blood pressure and syphilis.

PRACTICE OF COMPANIES IN SELECTION OF RISKS

In order to determine the principal variations in practice of the companies in their selection of risks, as shown by their Rating Schedules, I was courteously furnished with copies of such schedules by four of the largest companies, by two of the smaller companies, and by the Canadian Life Insurance Offices Association, the ratings of which are generally followed throughout Canada. The investigation might have included others but for the fact that many prominent companies use the Medical Impairment Ratings, with little or no change and their inclusion would not have added materially to our knowledge. It should be understood that while the companies under review expressed their opinions of risks in ratios of 100, such as 90, 110, 125, 150, etc. or as -10, +10, +25, +50, etc. not all of them used the numerical system. The adoption of the Medical Impairment Ratings or of any schedule of ratings does not imply that the numerical system is essential; in fact, the M.I.R. is available irrespective of the methods of medical selection.

To comment upon the differences found in the ratings of the companies for all the impairments would not be feasible. Accordingly the most significant in practice will be considered. In order to obtain an idea of the impairments which occur most frequently in companies issuing substandard policies, we have prepared a list according to the order of frequency in the New York Life. It has been found to agree closely with that of another company. Of course these impairments do not represent the same types as would occur in a company which limited its activities to standard insurance. Later this question will be discussed, but in the meantime the list of the more important impairments found in our practice is given:

Overweight

Heart murmurs

Abnormal blood pressure

- Family history of tuberculosis
- Alcoholic habits
- Albuminuria
- History of pulmonary tuberculosis
- Abnormal pulse rate
- Renal colic, calculus and gravel
- Gastric and duodenal ulcers
- History of syphilis
- Gall stones, biliary colic, infected gall bladder.

We shall now take up the impairments in the order of their importance according to the foregoing synopsis.

Overweight—There is much more uniformity of action with regard to overweights than twenty years ago. At one time many companies treated overweights lightly, not realizing the seriousness of the impairment at the middle and older ages. It is difficult to compare the schedules of the various companies as they are not prepared in the same way. One company has a basic build table which takes account of age and another which uses the relative weight at age 37. Nearly all make a difference in their mortality ratings according to height—usually in three groups, tall, medium or short. It would take too much space to make a comparison of the various companies' ratings in view of the diversities of treatment. The practical effect, however, may be seen by determining from the rules of several companies whether or not the procedure for men of medium height differs essentially with regard to granting policies on substandard or standard plans. Even such a comparison may not always be practical as one company may have a rule that, irrespective of rating, a standard policy may not be issued if the applicant be more than a certain percentage underweight or overweight.

Let us apply the foregoing test to applicants of medium height at 30% to 40% overweight, where there is said to be more difference in the practice of companies than at any other degree of over or underweight. A comparison of ratings from several companies for 30% overweight shows that, except at the younger ages, such a departure from the average weight would result in restriction to a substandard policy unless there were very favor-

able features to partly offset the overweight. Among persons 40% overweight our comparisons indicate that rated-up policies are usually issued. While the percentage to the total is not large, applicants of 30% to 40% at the middle and older ages are undoubtedly accepted by some companies at standard rates of premium but frequently are limited to Endowment Insurance maturing at age 60 or prior. Such exceptions are usually justified by the selector on the ground that the build is particularly good, that overweight is a family characteristic, that the applicant comes from a very long lived family or that "other companies are doing it".

Heart Troubles—Judging from the M.I.S. the majority of cases with heart involvements accepted by the companies, under sub-standard plans, consist of applicants with a constant systolic apex murmur transmitted to the left. The experience of the combined companies showed a mortality of 218% without hypertrophy (1,095 deaths) among the insured in occupations involving light work, the best class, if a history of an infectious disease were not included. Taking the seven Rating Schedules which are under consideration as typical of the action of most companies, the M.I.R. is generally followed with the exception of one prominent company which has not made any change in its ratings for several years and which has the unique practice of treating women with this impairment more severely than men. That company has had a mortality experience which justifies their action but is the result not primarily due to the greater difficulties of thoroughly examining women than men? It is interesting to note that many companies are now making a differentiation between persons engaged in clerical or light manual work and those doing heavy manual labor. Where there was also a history of rheumatism or chorea the M.I.R. was closely followed provided there was no hypertrophy; if there were even slight hypertrophy the practice of the companies is to decline the risk. Where the history was of an infection other than rheumatism or chorea the M.I.R. is usually followed, although two companies differentiated by age, being more severe at the younger than at the older ages at entry.

The next most important group of murmurs which came before the Joint Committee in point of number in its investigation of impairments (M.I.S.) consisted of applicants with systolic apex murmurs and mitral regurgitant murmurs which were not clearly defined, both as to transmission and constancy. Relatively few cases nowadays come within these categories as the companies endeavor to get an exact description of the heart. In the New York Life, for example, during the years 1925 to 1931 there were in these two groups only 1% of those in the four preceding classes. Accordingly ratings for these impairments need not be considered.

The next heart murmur in importance is a constant systolic apex murmur not transmitted to the left. Where there was no hypertrophy the mortality in substandard cases was found to be 156% (234 deaths). The companies seem to be following the recommendations of the Committee that the cases be taken as standard up to about age 40, treated as slightly substandard for ages 40 to 49 and rated +50 at ages 50 and over, with a tendency to be higher as age 60 is approached. The Committee's recommendations are evidently being followed with regard to those cases with hypertrophy or where there is doubt as to the character of the murmur.

In spite of the fact that there seem to be differences in the treatment of heart impairments by the various companies, judging by the number of appeals for lower ratings made by agents, there is not a wide variation in the Schedule Ratings of the companies under review. The differences doubtless occur largely from lack of uniformity in the reports as to the heart conditions by the medical examiners. Apart from that, the main point of difference arises with regard to murmurs which used to be classified as functional; should they be considered as standard or substandard? These appear nowadays as (a) a constant systolic apex murmur, not transmitted, (b) a similar basal murmur in the aortic area, (c) an inconstant systolic basal murmur in the aortic area, (d) an inconstant systolic apex murmur, and (e) a constant systolic basal murmur in the pulmonic area, not transmitted. The practice is to treat them as standard risks up to ages 35 or 40 and as slightly substandard risks thereafter except

as ages around 55 when $+50$ is fairly common. (The last two classifications of functional heart murmurs are rated a little more favorably than the first three mentioned.) It may be added that some of the companies granting standard insurance alone may be more liberal at the older ages than the companies rating up cases.

With regard to the differentiation in the ratings between cases with or without a history of an infection, it is believed by many authorities that heart murmurs are nearly always due to an infection and accordingly the higher ratings should usually apply. If it becomes known that the ratings are more severe where an infection is admitted by the applicant, there would be a predisposition to conceal the history. A suggestion has therefore been made that the companies treat all forms of infection, exclusive of acute rheumatic fever, the same as if there had not been a history of an infection, provided a favorable electrocardiogram had been obtained.

Little attention need be paid at this point to the other heart murmurs as they are generally declined or are rated severely.

In the M.I.R. no rating was given for palpitation of the heart. One prominent company provides a rating of $+20$ to $+50$ for a history of mild, occasional attacks occurring within five years, and no penalty after that time. Where the attacks have been frequent or severe that company charges at least $+100$ within 10 years or refuses to consider.

Blood Pressure—There was no subject which the Joint Committee on Mortality debated upon longer or analyzed more thoroughly than that of blood pressure. A distinct difference of opinion appeared between actuaries and medical directors. The statistical experience, which the actuaries are inclined to follow, indicated that a moderately elevated blood pressure was more serious than the medical directors are generally prepared to accept upon the basis of their clinical experience. As a result, the statistics in the M.I.S. were not followed by the Committee in preparing the M.I.R. but these ratings were distinctly more liberal than the experience indicated. For example, in the group of systolic blood pressure from 5 mm. to 15 mm. above the average the ratio of actual to expected deaths *among substandard*

lives was 153%, while that provided for in the M.I.R. was less than one-half of the extra mortality. Again, the extra mortality appearing in the M.I.S. for cases from 15 mm. to 25 mm. above the average was +81%, whereas the average provision in the M.I.R. was +30%. There were several reasons for this action of the Committee, the two principal being: first, that there were other factors which entered into the high mortality experienced and second, that ratings, based strictly on the M.I.S. were too high to be applied in practice, so that the companies adopting them would get only the poorer group of risks. The material of the New York Life, studied after the M.I.S. was published, proved the truth of the first as, among cases accepted on *sub-standard* plans which were from 5 mm. to 15 mm. above the average systolic pressure, over 40% showed a previous history of high blood pressure and in many other cases there was a history of albuminuria, glycosuria, abnormal pulse or heart murmur, these conditions having disappeared at the date of the last application. It should be emphasized, however, that investigations made by the New York Life and Metropolitan under cases issued as *standard lives*, indicated a definite increase in mortality with the departure from the average blood pressure. Cases in the group from 5 to 15 mm. above the average showed a relative mortality of 114% (481 deaths) in the New York Life experience, and 112% (991 deaths) in the Metropolitan; and in the group from 16 to 24 mm. departure above the average systolic the mortality was 128% (59 deaths) in the former as against 148% (133 deaths) in the latter. The ratings of the Joint Committee are accordingly more liberal than the experience of these two companies would justify.

As the subject is an important one we have obtained additional material regarding mortality according to the systolic blood pressure. The issues of the year 1923 were taken on policies issued by the New York Life at the regular rate of premium. The number of entrants among men was 159,000 with 4,491 deaths, and among women 31,000 entrants with 693 deaths. The material was combined, as the women represented only 16% of the total and as the basic table for measuring the expected deaths

included both men and women. The material has been divided into two groups of ages at entry:

ISSUES OF 1923 ON STANDARD LIVES CARRIED TO
ANNIVERSARIES IN 1932

Mortality by Policies

Expected Mortality Calculated by Company's Standard Table

| Departure from the Average Systolic Blood Pressure | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths |
|---|---------------------------|---|------------------------------|---|
| Mm. | Ages at Entry 15 to 39 | | Ages at Entry 40 and over | |
| -15 to - 5 | 750 | 93% | 739 | 85% |
| - 4 to + 4 | 1,199 | 97 | 819 | 100 |
| + 5 to +15 | 571 | 101 | 959 | 122 |
| +16 to +24 | 36 | 123 | 111 | 158 |

In a report on blood pressure made in 1925 by a Committee of the Actuarial Society it was stated that "The good effect of a low blood pressure seems to be greatest at the older ages at entry". That statement is confirmed by the foregoing investigation. Conversely a blood pressure higher than the normal shows a worse effect at the older ages than at the younger ages.

In order that comparisons may readily be made with the corresponding material of the two other investigations on standard lives to which reference has already been made, a synopsis for all ages together is now given:

| Departure from Average Systolic Blood Pressure Mm. | New York Life Issues of 1916 carried to 1930 anniversaries | | Metropolitan Issues of 1922 carried to Dec. 31, 1931 | | New York Life Issues of 1923 carried to 1932 anniversaries | |
|---|---|---|---|---|---|---|
| | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths |
| -15 to - 5 | 349 | 87% | 653 | 83% | 1,489 | 88% |
| - 4 to + 4 | 396 | 101 | 1,544 | 99 | 2,018 | 98 |
| + 5 to +15 | 481 | 114 | 991 | 112 | 1,530 | 114 |
| +16 to +24 | 59 | 128 | 133 | 148 | 147 | 147 |

The new experience confirms the previous experience of the Metropolitan and the New York Life to a remarkable extent.

When we are considering systolic blood pressures which are slightly above the average for the age should we not keep in mind that the lowest mortality is among the group of those 5 to

15 mm. under the average? If, for example, the systolic pressure of the applicant is 135 mm. at age 40 (10 mm. above the average) should we not consider such a case as one with a blood pressure of 20 to 25 mm. above the point of the best mortality?

As the subject of the relationship of the systolic to the diastolic blood pressure is a difficult one, we have prepared another investigation in the hope that it may be of value in clearing our minds. The data for the issues of 1923 in the New York Life have been carried to their 1932 anniversaries and have been classified according to the departure from the average systolic as well as the average diastolic blood pressure. This covers the same material as was used in determining the mortality according to departure from the average systolic pressure and applies to standard lives only. The expected deaths have been taken according to the company's standard table for the given period of exposure.

ISSUES OF 1923 ON STANDARD LIVES CARRIED TO
ANNIVERSARIES IN 1932

Departure from Average Diastolic Blood Pressure--Mm.

| Departure from Average Systolic Blood Pressure Mm. | -8 and less | | -7 to +2 | | +3 to +12 | | +13 and more | |
|--|------------------------|---|------------------------|---|------------------------|---|------------------------|---|
| | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths | Number of Deaths | Ratio of Actual to Expected Deaths |
| -15 to -5 | 418 | 89% | 884 | 87% | 180 | 93% | 7 | 90% |
| -4 to +4 | 191 | 99 | 1,149 | 97 | 637 | 101 | 40 | 87 |
| +5 to +15 | 58 | 124 | 439 | 103 | 860 | 115 | 166 | 134 |
| +16 to +24 | 1 | — | 18 | 128 | 73 | 132 | 53 | 189 |

In the group of +13 and more mm. departure from the average diastolic blood pressure, 84% of the data were at deviations of +13 to +17 mm.

The foregoing seems to indicate that a low systolic pressure gives a good mortality although the diastolic may be slightly above the average, whereas if the systolic is somewhat higher than the average the mortality increases with the departure from the average diastolic.

When the data were arranged according to deviation of pulse pressure from the average the mortality ratios did not seem to follow a clear line, but it was noted that pulse pressures below the average resulted in a mortality ratio of 108% while those above the average resulted in 129%.

The foregoing results should be studied in connection with those appearing in the Joint Committee's publication entitled "Blood Pressure" which appeared in 1925 and the paper of Mr. H. R. Bassford in October, 1932, to the Medical Directors Association, giving the experience of the Metropolitan Life Insurance Company. In order to save your time in studying these, I am presenting a table which gives a synopsis of the three researches. In each case a central figure has been used to represent each group of deviations from average blood pressure; thus +7 represented the group +3 to +10 mm.

SYNOPSIS—STANDARD DATA

Mortality Ratios to Standard Company Experience

All Ages Together

Deviations from Average

"Blood Pressure" (1925), Standard Men

(Number of Deaths) Ratio of Actual to Expected Deaths

| Systolic | Diastolic | -10 | 0 | +10 | +20 | Total Number of Deaths |
|----------|-------------|-------------|--------------|------------|-----|------------------------------|
| -20 | (850) 90% | — | — | — | — | 10,930 |
| -20 | (1,127) 96 | (1,506) 96% | — | — | — | |
| 0 | (1,094) 100 | (1,521) 100 | (2,089) 108% | — | — | |
| +10 | — | (773) 110 | (1,144) 124 | (313) 130% | — | |
| +20 | — | — | (465) 110 | (48) 150 | — | |

N. Y. Life, Standard Issues 1923

| | -12 | -2 | +8 | +15 |
|-----|-----------|------------|-----------|-----------|
| -10 | (418) 89% | (884) 87% | (180) 93% | (7) 90% |
| 0 | (191) 99 | (1,149) 97 | (637) 101 | (40) 87 |
| +10 | (58) 124 | (439) 103 | (860) 115 | (166) 134 |
| +20 | (1) — | (18) 128 | (73) 132 | (53) 189 |

5,174

Metropolitan Life, Standard Issues 1922, 23, 25, 26

| | -7 | 0 | +7 | +15 |
|-----|-----------|-----------|------------|------------|
| -10 | (585) 81% | (133) 91% | (82) 100% | — |
| 0 | (656) 105 | (533) 92 | (719) 99 | (98) 109% |
| +10 | (262) 114 | (280) 107 | (550) 107 | (184) 120 |
| +20 | (20) 129 | (22) 132 | (90) 171 | (56) 158 |

4,270

A study of this table shows that the three sets of data are in general agreement.

A table arranged by age-groups (which to save space is not published) showed that the Metropolitan Life and New York Life at entry ages 40 and over had a distinctly higher mortality than at ages under 40. This characteristic did not appear in the older data of "Blood Pressure" (1925).

With regard to the ratings for blood pressure, two of the large companies under review and the Canadian companies have adopted the M.I.R., while another of the largest companies has ratings which increase more rapidly than the M.I.R. as the departure from the normal systolic increases. For example, those in the group-age 30 to 39 have a rating at 145 mm. of +15 against that of the Joint Committee of +30, at 150 mm. of +50 against +45 and at 155 mm. of +100 against +65. As already stated, a number of companies consider the ratings of the Committee too lenient.

With reference to diastolic pressure the companies under analysis have generally followed the recommendations of the Joint Committee, except that there is a tendency to stiffen the ratings for the higher diastolics. Unfortunately the companies have not adopted a uniform practice with regard to taking the diastolic either at the fourth or at the fifth phase. Even in a company which instructs its examiners to adopt the fifth phase there is evidence that the younger examiners are using the fourth phase, which is taught in many medical schools. The best way to meet this difficulty would be to call for the diastolic reading at both the fourth and the fifth phases.

So far as concerns low systolic pressure, at least two prominent companies consider that the M.I.R. is too liberal. Their ratings, for example, for a systolic pressure of from 30 mm. to 40 mm. below the average are from +30 to +50 against the Committee's ratings of from +15 to +30.

Tuberculosis in the Family History—Before making any comparison of the ratings of the different companies it might be well to review briefly a few of the conclusions appearing in the Supplement to the Medical Impairment Study. The Committee

stated that the ratings of the companies at the time of their investigations (1930) were inadequate at ages under 30 for those of average weight or overweight; "in fact at those ages there was an increasing divergence of the average company rating from the experience as the number of cases of tuberculosis in the family history increased." A statement was also made that at ages 45 and over the number of cases of tuberculosis in the family history had apparently little effect on the mortality experience. It is hardly necessary to again mention that age is, for this impairment, a more important factor than build. But it may be of value to emphasize that although there had been a great reduction in the proportion of deaths from tuberculosis among insured lives there had been a marked improvement in the general mortality so that there was not much reduction in the *relative* mortality rate in the tubercular family groups. Such a result was contrary to the prevailing impression that, with the reduction in the death rate from tuberculosis, there would be a decreasing mortality relative to the normal among persons who had a family record of tuberculosis.

The mortality was so high in the M.I.S. among persons of light weight at the younger ages with three or more deaths from tuberculosis that the Committee did not follow the experience in suggesting a schedule. It was thought that, with the increase in knowledge, greater care would be taken than formerly in the selection of applicants who had a strong tubercular family history.

A comparison of the M.I.R. with the ratings of the companies indicates a disposition to follow the suggestions of the Committee. One company, however, has higher ratings among the underweights but more lenient among those slightly overweight, while another prominent company is slightly more lenient than the M.I.R. at the younger ages only. The fact that a few companies have been more liberal than indicated by the M.I.R. with three or more cases of tuberculosis in the family history, is of little significance as the percentage of cases of this type is small, and as they are not treated as substandard risks except at the younger ages.

Habits as to Alcohol—The preparation of a schedule of ratings for persons who use alcohol freely or who used it immoderately in the past is a very difficult matter. One of the largest companies does not attempt to make any rulings but leaves the decision in each individual case to the medical staff. As that process must result in different offers to applicants under practically similar conditions, other companies have attempted to lay down a program which would make the ratings fairly homogeneous. These ratings are intended to represent an average under the conditions presented, with the expectation that variations may be made either toward leniency or severity as the individual departs from the average of his type.

In comparing the Schedules of several of the large companies with the M.I.R. there is a noticeable tendency to be a little more liberal when there has been an immoderate use of alcohol a very few times a year and to be distinctly more severe when there has been such use occurring as often as once a month. It is quite clear from a study of these ratings that the companies are averse to insuring on any terms any one who becomes intoxicated two or three times a month, or who goes on protracted sprees even though these occur seldom. If I may endeavor to read the minds of the Medical Directors of many companies they are disposed to consider that addiction to alcohol is likely to increase rather than to decrease in an individual who has formed the habit and that the so-called cures are not so effective as their advocates aver.

When it comes to a steady use of alcohol, such as two or three glasses of whiskey a day, the recommendations of the Committee are considered reasonable, but the provision for extra mortality is seemingly based on the belief that the individual's consumption is understated or that it is likely to increase in the future.

With reference to those who have taken a cure for alcoholic habits the recommendations of the Committee find favor, largely due to the fact that there is no other extensive material upon which to base our practice.

The acceptance of the ratings for this impairment by many companies may be largely based on the well founded belief that

there was no subject, except possibly blood pressure, on which a committee of experienced medical directors and actuaries spent so much time in an endeavor to arrive at conclusions which would be just to the applicants and to the companies.

Albumin—In the case of a trace of intermittent albumin the practice of the companies is in line with the recommendations of the Committee although one large company is more severe where the trace is approximately .05%, treating all such cases as substandard risks. Where the amount of intermittent albumin is moderate or large the companies whose ratings have been reviewed follow the M.I.R. or are more severe. If there be a constant trace of albumin these companies have adopted the Committee's suggestions, except one which is more lenient. With regard to a moderate amount of constant albumin, one company declines all cases where the proportion of albumin on examination is .05% or more, while the others follow the M.I.R.

Pulmonary Tuberculosis, History of—In the M.I.R. there are three subdivisions of pulmonary tuberculosis: (a) not specified as to physical signs or bacilli, (b) with physical signs on examination and (c) without physical signs on examination. The significant features of the M.I.S. are that the mortality was high in the early years following the attack, particularly among underweights, that the mortality among those accepted by the companies after ten years had elapsed was "standard", and that the death rate in the three classes from tuberculosis of the lungs was from four to five times the normal. The number of cases accepted by the companies even after ten years from the date of the attack was very small comparatively. The M.I.R. indicates that, in the belief of the members of the Committee, insurance can be issued with caution at standard rates to persons distinctly overweight at the older ages at entry a few years after the attack, and may be granted even at ages around 35 after the lapse of ten years, if the applicant is of average weight or more. This program is acceptable in the main to the companies whose ratings have been analyzed, except that one of them is inclined to be more liberal at the younger ages while another company is more severe at practically all points. There seems

to be a tendency to treat cases alike whether there is the presence or absence of physical signs in old cases of tuberculosis, on the ground that the finding of such signs depends largely upon the ability of the individual examiner.

Pulse Abnormalities—In order of frequency, irregularities of the pulse rate stand eighth in our experience on substandard risks. The companies seem to follow the M.I.R. although one company has a definite rating of +50 for a persistently rapid pulse from 90 to 100 and another does not take youngsters with a pulse rate over 94 per minute. Where the pulse rate is persistently above 100 beats per minute the almost universal practice is to decline to grant insurance on any terms.

A persistently low pulse is generally considered by the companies as a good sign provided it is not under 55, but one company evidently wishes to be conservative as it imposes +25 for a pulse rate of 55 per minute. If the pulse rate were 50 the same company would rate a case at least +35, whereas other companies would accept as standard, everything else being satisfactory. A pulse distinctly under 50 per minute appears to be considered cause for declination or for a substandard rating since it is frequently associated with a fatty heart or with myocardial changes.

A discussion of the treatment of an intermittent pulse is not feasible as it would call for too many details—the ratings depend on so many factors. Variations in ratings are made according to whether the intermittency disappears, persists or increases on exercise and whether there has been a favorable electrocardiogram. Age is also a factor. The ratings of the Joint Committee are usually followed; in fact several of the large companies have adopted them unchanged. The other companies, which have approximately the same ratings, also accept the M.I.R. in considering that the hazard increases with advancing age; thus, a case which might be rated +60 if the applicant were age 35 would be declined under exactly the same conditions if he were age 55.

In the case of an irregular pulse the companies under consideration treat it the same as a corresponding intermittent pulse,

with the exception of one company which is so severe that it declines to consider an irregular pulse rate of even 5 per minute, although granting policies to under-average risks.

In the M.I.R. a history of paroxysmal tachycardia is not mentioned; neither are any ratings provided in any but one of the schedules in my possession. It may be interesting, therefore, to give roughly the ratings of that company. When there had been one attack within a year the ratings run from +75 to +200 depending upon the age. A history of an attack 3 years ago would have a rating of +35 if the applicant were under age 30, running up to +75 if he were over age 40. When there had been more than one attack the company does not accept the applicant within the first year, and at the end of 5 years would rate as high as +50 at the young and +100 at the middle ages.

Renal Colic, Calculus or Gravel—My comments may be more clear if references are first made to the M.I.S. In reviewing this class the Joint Committee stated that the operated cases had a worse experience upon the whole than the non-operated. Under either condition they recommend that the cases be not accepted on any terms if a kidney stone be present at time of examination. If none be found, the non-operated cases would be considered as slightly substandard during one year after an attack and thereafter rated to an extent which would permit the risk to be accepted standard if otherwise first class. When there had been two or more attacks without operation the case is apparently not considered so serious as if an operation had taken place. The Committee recommended that, without the presence of a stone, the operated cases should be declined for at least a year and should be considered distinctly substandard for ten years following the operation.

The foregoing practice is generally followed by the companies under consideration, although one prominent company apparently accepts cases where a stone is now present; they are treated as distinctly substandard for several years after the attack. The same company also presents a schedule for three or more attacks separate from that for two attacks. The Committee by expressing the mortality rates as a range had left this matter to the

discretion of the individual medical officer. A medical director suggests that in the case of renal colic with an operation a more liberal rating might be granted if the pelvis of the kidney only had been opened.

Gastric and Duodenal Ulcers—It seems best to consider at the same time these two types of ulcer. The Joint Committee felt that the hazard was a decreasing one and provided for a series of extra premiums decreasing with the time elapsed since the attack or the operation. The M.I.R. for gastric ulcer without operation has been adopted by the companies whose schedules are being analysed, with two exceptions, both more severe—a large company which does not differentiate between one and two attacks and a smaller company which charges a stiffer extra premium for one attack of gastric ulcer without operation, declining persons who have had two attacks even though a considerable time has elapsed since the last attack.

Where there has been an operation for gastric ulcer the companies follow the recommendations of the Committee with the exception of one large company, to which reference has been made, whose ratings are more severe for several years following the operation.

Until a few years ago the rating for a duodenal ulcer without operation was more liberal than for one with an operation, on the assumption that an ulcer was usually less serious if an operation had not been performed. With the trend towards medical treatment in the types of cases where operations had previously been performed, the companies began to consider both types alike. In the opinion of the Joint Committee the rating for an applicant with a history of one operation for duodenal ulcer would be the same as if he had not had an operation but had been treated medically. Based on the favorable experience at the Mayo Clinic, published in 1919, where there seems to be no appreciable hazard after the second year following the operation, a number of companies charged an extra premium for two years only. Within a few years thereafter, however, the treatment of many companies became more severe than was indicated by an analysis of the Mayo Clinic results, partly due

to their own experience and partly due to the growing conviction that the results were not always so favorable as had been obtained by the Mayos.

The practice of the companies under review indicates that they are largely adopting the recommendations in the M.I.R. with regard to duodenal ulcers, namely, to defer consideration for one year following the operation or following one attack and to charge an extra premium for the second year of \$5.00 during 4 years, for the third year of \$5.00 for three years and so forth. Two companies, however, are more severe, deferring any action until after two years, but only one is slightly more severe than the M.I.R. thereafter. So far as two attacks are concerned the practice is in line with the Committee's suggestion except for one company which makes no differentiation between one or two attacks, but that may be changed after a review now in progress.

History of Syphilis—Where there is a history of syphilis which does not appear to be "cured" and has not had thorough treatment, the companies decline to consider the risk on any terms although an occasional case may be accepted on a very conservative basis. That is also the procedure when the applicant is designated as "not cured" but is said to have had thorough treatment. The principal types which come before us are those which are said to be "cured", which is defined to be "freedom from any active symptoms of the disease as indicated by two or more negative Wasserman tests or a negative spinal fluid test". For a number of years this type, if there had been thorough treatment, was considered as borderline or slightly sub-standard; in fact, several companies issued 15 and 20-Year Endowment policies maturing usually before age 60 at regular premiums. This was done on either of two assumptions (a) that the extra risk decreased with advancing age and with the period elapsed since the beginning of the history, or (b) that Endowment insurances carry a higher mortality than Ordinary Life policies. From my observation, this practice has largely changed so that most companies charge an extra premium or advance the age, except a few companies which endeavor to

select only the very best of such risks, charging the regular premium. The present change commenced with the publication of the Medico-Actuarial Investigation, the results of which were confirmed by the M.I.S. Many Medical Directors had hoped that, with the new Salvarsan treatment, there would be a lessening of the mortality from this disease but any favorable effects in relation to insurance mortality as a whole are not yet evident. It is possible that the more recent forms of medical treatment have tended to defer the more virulent effects of the disease such as paresis and locomotor-ataxia.

All of the companies whose practice is under review agree substantially with the M.I.R. One large company, however, is more severe in rating the best cases and more lenient with the worst cases of those who are "cured whether there appeared to be thorough treatment or not".

Gallstones, biliary colic and infected gall bladder—In the M.I.S. the Committee made three subdivisions of this type of impairment (a) gallstones or infected gall bladder without an operation, to which they added biliary colic not clearly coded elsewhere; (b) removal of gall bladder, whether due to stones or an infection, without stones; and (c) drainage of the gall bladder for gallstones or for an infection without stones. In commenting upon these groups the Committee stated that "the mortality among cases treated either by medicine or by removal was approximately normal", but among cases where drainage was recorded it was about 40% in excess of the normal. An analysis shows that in these three subdivisions the practice of the companies is in harmony with the recommendations of the Committee, except that one large company is more lenient for either one or two attacks of gallstones without operation. The general practice in the case of one attack of gallstones without operation is to decline for one year and then to charge an extra premium of \$5.00 per \$1,000 for four years thereafter. Where there are two attacks the case is deferred for two years and a larger extra premium charged for the ensuing four years. In the case of the removal of the gall bladder the Committee recommend an extra premium of \$5.00 during the second year following the removal, which

appears to be considered reasonable although one or two companies in our analysis are more lenient, charging the same extra premium from the 7th month, instead of refusing to grant insurance.

The other types need not be discussed in detail as an infected gall bladder without operation or with surgical drainage is usually treated the same as gallstones without operation, while the removal of an infected gall bladder, without stones being found, is considered on the same basis as the removal of the gall bladder for gallstones.

BORDERLINE RISKS

A study which has apparently not yet been made is that of analysing the types of impairments which are looked upon as borderline by the companies that do not issue policies on substandard plans. Some of the necessary material is available through a review of the agents' manuals. Four of these have been obtained from the most prominent companies supplying their agents with a statement of the principal impairments which would be declined or might be considered as borderline. If the latter, the cases may be submitted to the home office with the understanding that the most favorable might be accepted and the others declined. In the following, reference is not always made to four companies as sometimes only two or three deal with the impairment:

Albuminuria—This is not mentioned in two of the four manuals of instructions to agents, while in the other two it is considered borderline, if intermittent.

Appendicitis without operation—While one company considers that a single attack of appendicitis within a year is a borderline risk, another declines such business until three years have elapsed. It is evident, however, that in general companies do not consider the case as substandard after one year has elapsed since the operation.

Blindness—In the case of this defect two companies do not accept on any terms, whether the blindness affected one or both

eyes, while another is willing to accept the best of the cases at the regular rate of premium, even if totally blind.

Personal History of Tuberculosis of the Lungs—Two of the companies decline to consider applicants irrespective of the time elapsed since the date of the attack, but the other two companies believe that if eleven or more years have elapsed it is a borderline case provided the build and family history are good and an X-ray favorable. The same applies to tuberculosis of other parts, such as bones.

Cure for Alcoholic Habits—The practice of these companies is to decline until several years have elapsed after the cure for alcoholism was taken. Acceptance would then be contingent on total abstinence.

Deafness—Treatment by the four companies is generally "standard" if only one ear is affected and "borderline" if both ears.

Gallstones—The practice is to treat a history of gallstones without operation, whether drainage has been employed or not, as making the applicant not acceptable until several years have elapsed since the attack, after which time the case is borderline. A more lenient practice applies if the gall-bladder has been removed.

Hernia—It is interesting to note that the practice ranges for a reducible hernia without truss from standard insurance to a declination, but I question whether the strictest company adheres to its rules in many cases.

Pleurisy—A history of pleurisy within recent years places the applicant in the borderline category, although two companies do not accept until at least five years have elapsed.

Renal Colic, with or without operation—The most liberal of the four companies believes that a case of renal colic should be treated as borderline from the seventh to the twelfth months after one attack or operation, and thereafter be acceptable at the regular rates, while the other companies require that from one to five years must elapse before the applicants be considered as standard risks.

Duodenal Ulcers—Where there has been one attack without an operation the cases are declined for from three to five years and

looked upon as borderline for at least two years thereafter. In general, it may be said that where there has been a duodenal ulcer, whether with or without operation, the cases are not granted insurance by these four companies at the regular premium rates for five years on the average, although the very best of them may be acceptable after three years.

The four companies now under review generally decline applicants with histories of syphilis or of organic heart disease.

While the foregoing indicates the principal types of borderline cases, exclusive of those due to build, family history or blood pressure it does not show them in the order of their frequency and such information is not available to me. The Medical Director, however, of one of the large companies restricting its coverage to standard risks, informs me that the *principal* types calling for special attention as borderline risks are the following:

Overweight

Underweight

Underweight with family history of tuberculosis

Unfavorable family history

Abnormal blood pressure

Overweight, not excessive, with blood pressure in the upper reaches of the normal limits

Favorable types of heart murmurs

Underweight with chronic infection of the upper respiratory tracts.

CONCLUSION

There were a number of interesting comments made in reply to my letters of inquiry. One of them was to the effect that a medical director could not disregard the conclusions in the M.I.R. unless he had reliable experience of his own company or had particular knowledge of certain impairments. Doubtless many of you will agree with this point of view. Some medical directors, however, especially in companies which limit their business to standard risks, are more inclined to follow the tradition of their company and their individual knowledge, including a limited mortality experience of their own company on the ground that

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the M.I.S. did not reflect the conditions under which their selection was made. While sympathizing with them, there is undoubted value for every one in such investigations as the M.I.S. and in the composite opinions (M.I.R.) of a committee of medical directors and actuaries.

By writing this paper I had hoped to acquire a large amount of information with regard to the practices of other companies in the selection of risks as shown by their attitude towards the M.I.R. Such information will be put at the disposal of the Joint Committee on Mortality in the event that they desire to add to the present ratings among impairments now in the M.I.R., to modify them at a few points, to introduce some of the more recent designations of disease or to include ratings for very unusual impairments.

In conclusion, my studies indicate that the Medical Impairment Ratings are used very largely by companies which grant business on both standard and sub-standard plans, but to a less extent by companies limiting their transactions to the former; that they have tended to produce more uniformity in the action of the companies and that they have caused many actuaries and medical directors to give a more intensive study to individual impairments, especially where their inclination was to differ with the Joint Committee.

DR. MUHLBERG—I assume Dr. Hunter very courteously asked me to discuss his contribution, principally because he desired an expression of opinion from a Medical Director of a medium-sized company. Besides, having served as a member of the Medico-Actuarial Committee, I was in a position to listen to the discussions of the other members of the Committee and consequently, to become acquainted with some of the complexities of the work.

The Company I represent is one that uses the Medical Impairment Ratings to the extent of 85% or more, because I feel that, while these ratings are in many instances compromises, they represent the best opinions and practices of Medical Directors and Actuaries who have had the widest experience in dealing with medical impairments, particularly those that call for substandard

treatment. Dr. Hunter, of course, was our inspiration and guide and performed the major part of the work. It was really a Herculean labor.

Medical Insurance Selection is not essentially different from medical practice. Medicine is partly a science and partly an art, and any doctor who practices only the art does not make use of all weapons at his command. So any Medical Director who does not avail himself of the Medical Impairment Ratings or the material in the Medical Impairment Study is neglecting the scientific assistance that these studies offer. The Medical Impairment Rating booklet embodies our science, as opposed to individual clinical selection, that represents our art.

That the Committee recognized this distinction is evident in the fact that the Medical Impairment Ratings do not always coincide with the actual experience expressed in the Medical Impairment Study. As an example, Dr. Hunter acknowledges that the blood pressure ratings are rather liberal, in view of the Medical Impairment Study experience.

It is probably entirely conservative for companies doing a large volume of business to operate almost solely with the Medical Impairment Ratings recommendations, combined with the Numerical Rating method, because the law of average will smooth out any discrepancies; but I believe that even the larger companies use individual selection. For smaller companies, where meagre volume may cause marked variations in the curve of experience, individual selection is still very important, provided the Medical Impairment Ratings are used as the basis for such departures from the average. That careful medical selection has markedly influenced the death rates in many impairments is quite evident when one compares the tables in the Medical Impairment Study giving the standard and substandard experiences. In some instances, the mortality figures are markedly divergent. This may be explained either by a lack of homogeneity in the substandard cases, or a difference in degree of the impairment, or by the severity of selection.

Nor is it incumbent on all of us to accept the Medical Impairment Ratings recommendations as laws of the Medes and

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Persians. There will, no doubt, be further revisions in the future. In fact, Dr. Hunter's paper gives excellent examples of the testing of the ratings, and similar researches will no doubt absorb much of our energies in the future.

Nor need all us subscribe to every item, even in the absence of further research. Personally—and I am expressing solely my own opinion—I do not concur in the recommendations of the Joint Committee under 309—Basal Murmur, aortic area, systolic constant, not transmitted. A systolic aortic murmur is, in my opinion, so often an expression of aortic valve disease, or aortic sclerosis and dilation, that it is hazardous ever to view it as functional. I believe that the favorable experience in the group was largely due to the intrusion of material that should belong in 316—Basal Systolic Pulmonic Murmur, which is recognized by cardiologists as being the commonest of heart murmurs and practically always functional. Many examiners do not auscultate at the second left cartilage, with the result that a diffused pulmonic systolic murmur is often diagnosed as a slight systolic aortic.

Dr. Hunter states that where companies have reliable figures on any particular impairment which contradict the ratings of the M.I.R., such companies are warranted in following their own experience. My own Company has made a study of the significance of casts, and we are fairly convinced that applicants showing a moderate number of casts are standard risks for five or ten years and may be offered modified policies to expire in five or ten years. But I believe that it might not be wise for another company to follow our practice unless the same technique and the same standards for diagnosing casts are used.

I believe that the blood pressure ratings are essentially correct, provided, particularly in the higher brackets, one is sure that there has been no chiseling. Dr. Hunter's recommendation that we request our examiners to take the diastolic at the fourth and also fifth phase is an excellent one, but I believe we ought likewise insist on knowing whether any other blood pressure estimations made by the examiner gave figures different from those stated on the examination blank.

Applicants, agents and examiners are becoming increasingly blood-pressure-conscious. Where the blood pressure is found abnormal on first examination, too often other examinations are made under conditions not fair to the insurance companies, and we are given the favorable readings without any mention of the unfavorable ones. This, I believe, may possibly explain the sharp up-curve in mortality experience for applicants with blood pressures $+16$ to $+24$ for issues of 1923, as compared with issues of 1916. The 1916 blood pressures presumably indicated more nearly the average blood pressure, uninfluenced by manipulation.

There are some considerations to which we Medical Directors must pay heed. The mortality ratio of any group is by no means a fixed percentage. New methods of medical diagnosis and treatment, changes in economic or environmental conditions, markedly influence our experiences. I need merely cite the fact that each succeeding mortality table differs from every previous one, and certain diseases, such as tuberculosis, show a progressively declining mortality rate, to be replaced later by apparent increases in heart and cancer deaths. And as we advance in medical diagnosis, our practices must be determined upon or modified long before we have an actual experience to guide us. This is going on at the present time with electrocardiographic interpretations. Such considerations may warrant our modifying the ratings in some instances, although this factor was often discounted by the Committee in determining the ratings.

Many Medical Directors have expressed the fear that a slavish adherence to Medico-Actuarial investigations leads to excessive standardization, and that the Medical Director will ultimately be replaced by the mechanical operations of the lay medical underwriters. I do not believe we need fear any such eventuality. On the contrary, the newer methods of medical diagnosis are becoming so highly technical that even now we need expert advice by specialists outside of our office, and not infrequently it puts considerable strain on our medical knowledge to evaluate the interpretations of these experts.

I was much interested in Dr. Hunter's remarks on palpitation of the heart. It is recognized by cardiologists that the earliest

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evidences of myocardosis are subjective and usually occur without any demonstrable abnormalities in physical signs or electrocardiographic determinations. A history of palpitation of the heart in men past age forty, combined with increasing shortness of breath and twinges of pain near the præcordium, should excite a suspicion of myocardial degeneration. As a rule, we can not elicit past symptoms of slight pain or progressive dyspnœa, but palpitation of the heart is often so alarming that an applicant will acknowledge it. It might be well to stress this impairment in our examination blanks, if for no other reason than to supply data for statistical investigation later on.

The essayist has I believe very tactfully refrained from commenting on glycosuria and its significance. This impairment is perhaps as difficult to evaluate as alcoholic habits; besides, we know at present so little of the trustworthiness of our special blood tests. It would, therefore, be advisable for the Committee to recommend ratings more detailed than those suggested in the M.I.R.

History of Tuberculosis.—It is hoped that in the future we may be able to refine our ratings so that the difference in the tuberculosis rate between the sexes may be given consideration. Dr. Hunter, in a paper entitled "Tuberculosis—A Review", presented before the Ninth International Congress of Actuaries, graphically indicated this difference in the trend of mortality and his conclusions are being confirmed by more recent studies.

The peak in the tuberculosis mortality, especially for men engaged in manual labor, and dwelling in cities, is reached at age fifty or fifty-five, while for females, the maximum rate is in the twenties. In fact, tuberculosis claims more male victims per 100,000 between ages fifty to sixty and between sixty and seventy than between ages twenty and thirty. This difference in sex must be remembered in dealing with those predisposed to tuberculosis by reason of previous history, or family history, or exposure to infection, or underweight.

As Dr. Hunter indicated very frankly in discussing "Habits as to Alcohol", there was no subject, except possibly blood pressure, on which the Medico-Actuarial Committee spent so much

time in an endeavor to arrive at conclusions that would be just to the applicants and to the companies. It is evident that the recommendations made by the Committee represent a compromise of varied opinions, and in consequence, ratings were made that permit of wide latitude for individual selection and opinion.

I need not comment upon the other impairments discussed by Dr. Hunter. I am so much in agreement with his conclusions, that my remarks would be merely a repetition of his analysis.

It might be of some interest to Medical Directors of companies that are in the same class for volume of business, etc., as the company I represent, and that are not now engaged in writing substandard business, to know that somewhat timorously, we launched into this field in 1922. At that time, we had no such assistance as the Medical Impairment Ratings offer and there was some doubt as to the correctness of ratings then available. Since then many changes have been made in the appraisal of impairments. But in spite of this, our substandard mortality experience has been very satisfactory, and notwithstanding our small volume, has followed closely the predictions of our tables for the various classes of substandard issues. Our substandard business has also demonstrated its value to our applicants and has, after a short period of doubt on the part of the agency staff, been highly appreciated by the underwriter in the field; and, perhaps most pleasing of all, has made it easier for the Medical Department. Disputes between the Medical Department and agents arise mostly over risks that are just over the borderline for standard approval. Agents are usually successful, without further argument, in delivering policies with slight ratings. Neither the company nor the Medical Department, nor the agents would today want us to abandon the issuance of substandard policies.

Members of the Joint Medico-Actuarial Committee, and particularly Dr. Hunter, are to be congratulated that so many companies have adopted the Medical Impairment Ratings. It certainly ought to make our essayist feel that his labors, though tremendously burdensome, are being rewarded.

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DR. SCADDING—The discussion will be continued by Mr. John G. Parker, Actuary of the Imperial Life Assurance Company of Toronto. Mr. Parker has the rather unusual distinction of being a Fellow of the Actuarial Society of America, the American Institute of Actuaries and the Institute of Actuaries of England. He is now Vice President of the Actuarial Society of America, Chairman of the Joint Examination Committee of the Society and Institute, and a former president of the American Institute of Actuaries.

MR. PARKER—One of the most important advantages of any system of medical impairment ratings is that by its general adoption it automatically produces a greater measure of uniformity in the selection of standard and substandard risks. In this paper, Dr. Hunter, by showing the departure from the M.I.R. in the practice of various companies, has contributed a great deal to securing a still greater measure of uniformity amongst the companies who have adopted, or partially adopted, these ratings.

The Canadian companies have always shared with our cousins across the line the belief that competition in the terms offered to borderline or substandard risks has been productive of loss to the competing companies.

A good many years ago, 1919 in fact, a committee of Medical Directors of the companies in Canada, assisted by a few of the Actuaries, worked steadily through a long series of weekly and semi-weekly meetings and evolved a schedule of ratings which were published through the courtesy of the Canadian Life Insurance Officers' Association. This has been revised at various times, the last publication of the rating sheets being in November, 1930. Since that time the Committee, with some changes in its personnel but still under the able chairmanship of Dr. Scadding, has adopted the Medical Impairment Ratings as published by the Joint Committee under the chairmanship of Dr. Hunter.

Naturally the Committee in Canada in studying the M.I.R. could not agree in entirety with the ratings proposed. There was, however, the feeling that the adoption of these ratings

would give the companies a greater knowledge of their virtues and their faults so that when the time came for re-publication practical suggestions could be offered as to necessary amendments.

Although the Canadian Committee have recommended the general adoption of the M.I.R. yet I do not believe all of the companies have discarded the ratings of the Life Officers' Association. I will discuss briefly some of the more important exceptions and some of the suggestions which have been offered.

Syphilis—The Canadian companies feel that the ratings for those cases coming under the category, "Not thorough treatment" are not sufficiently high and we would prefer an increase in such ratings.

Tuberculosis of the Bone or Joint—While the Canadian companies are following the suggestion to rate the above impairment the same as "Pulmonary tuberculosis, without physical signs", yet I think there would be greater unanimity in practice if the rate were about two-thirds of that imposed for this latter impairment.

Chorea—It is generally thought that a history of chorea should be dealt with exactly the same as a history of acute articular rheumatism.

Neurasthenia—It is felt that where there have been two or more attacks the ratings proposed are too lenient. It has also been pointed out to me that it is difficult to get uniformity in any particular case due to the fact that the definition of "mild" and "severe" attacks and the number of attacks is frequently a matter of judgment.

Apex Murmur, Systolic—In such impairments as "Apex Murmur, Systolic, Constant, with History of Rheumatism", some of the Canadian companies have stated that a six to ten year waiting period after the rheumatism is longer than they can possibly impose in the actual conduct of their business in the field. As the ratings proposed are, on the whole, higher than originally suggested in the ratings of the Canadian Committee, I am not sure that they have been as generally adopted as the ratings for other impairments.

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Persistent Intermittent Pulse—It has been difficult to secure uniformity of action in regard to this impairment, due to the wide range of impairments which might be covered by this particular heading. Our Committee feels that some companies are accepting for standard insurance a fair number of extra systoles which disappear on exercise and there is the general impression that there is considerable lack of uniformity in dealing with this impairment.

Blood Pressure—There has been some difficulty in getting the companies to adopt the ratings for blood pressure although as a practical matter the old ratings put out by the Canadian Committee differ little in results.

In the M.I.R. no rule is given in regard to a past history of high blood pressure. The Canadian Committee feels that the history should be taken into account by means of a formula which would give greater weight to the higher readings. One proposal was to multiply the highest reading by 5 and the two most recent lower readings by 3 and 2, dividing the total by 10, to obtain the reading with which to enter the table.

The Committee further feels that diastolic readings over 100 generally render the risk uninsurable.

It is also felt that an applicant with a systolic blood pressure of 145—150 accompanied by a diastolic of 80-85 is probably a better risk than the one with the same systolic blood pressure but with a diastolic between 95-100. The M.I.R. would, however, rate these risks the same.

Pleurisy—Some companies are finding it difficult to make the marked distinction between "dry pleurisy of less than two weeks duration" and "more than two weeks duration", which the M.I.R. suggest.

It is also felt that it is rather severe in actual practice to treat "pleurisy with effusion" the same as "Pulmonary tuberculosis without physical signs".

Glycosuria—It has been pointed out that uniformity cannot exist in the group of impairments under this heading when different companies use different requirements for tolerance tests and blood sugar estimations. The Canadian Committee feel

that the instructions which they published in the Bulletin of the Life Officers Association were of distinct value in securing uniformity in the tests required of the applicant during examination and in the final rating of the risk.

Hæmaturia, etc.—It has been difficult to get uniformity with regard to the rating of cases showing a few blood cells or a few pus cells when found in every sample submitted.

Renal Colic—A considerable lack of uniformity might arise due to the classification "with operation". Some companies feel that if a simple operation has been performed, not a "nephrotomy" that the ratings are rather severe.

The Canadian Committee feels, while they have recommended the companies to follow the ratings in the M.I.R., yet the old method of extra premiums for a few years is more practical.

The Use of Alcohol—The greatest difficulty has been found in obtaining uniform decisions where there is a history of the use of alcohol. In the first place it is felt that there is little chance of placing a rated policy in the good substandard cases. The offering of a rating in such a case is tantamount to declination. Consequently, the companies find it difficult to place the policies where the moderate ratings are imposed, say from +40 to +75. Policies with the higher ratings may be easier to place but, on the other hand, the risks are extremely hazardous. There is also a decided lack of uniformity under this group due to the difficulty of obtaining accurate information.

Goitre—It is felt that there is a lack of uniformity in decisions on cases showing a history of goitre. Several reasons are given for this. It is frequently difficult to obtain information, or else such information is not available as would enable us to definitely classify the goitre. Some companies take note of time of convalescence, others do not. There is also a marked difference of opinion in companies as to the seriousness of goitre.

In conclusion I would like to add that the publication of the M.I.R., in my opinion, will have done more to produce uniformity of selection than any other thing. In order, however, to keep such uniformity it is essential that the Joint Committee have in mind the necessity for constant revision of the ratings

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and the consequent republication of the M.I.R. at periodic intervals. It might be possible for the Joint Committee to prepare amended ratings whenever a different decision is reached in regard to some important impairment. These could then be forwarded to the companies and this might obviate the necessity of re-publication of the entire book.

I would like to take this opportunity of expressing the thanks for the Canadian companies for the paper which Dr. Hunter has submitted to this meeting and also for the splendid contribution to the good of the life insurance business in the preparation of the ratings submitted to the companies in the M.I.R.

FURTHER MORTALITY DATA OF NON-ORGANIC SYSTOLIC HEART MURMURS

By J. W. FISHER, M. D.

Medical Director, The Northwestern Mutual Life Insurance Company

In 1905 the Northwestern Mutual Life Insurance Company began accepting carefully chosen risks with non-organic systolic murmurs. In separating non-organic from organic murmurs the following restrictions governed our selection:

1. Systolic in time, but heard either at apex or at base.
2. No diastolic murmurs (including pre-systolic of any kind.
3. Not transmitted.
4. Apex beat in normal area.
5. No other evidence of hypertrophy.
6. No accentuation of second pulmonic tone.
7. No abnormality of pulse rate or rhythm.
8. Arterial tension normal.
9. Reaction of heart rate to exercise normal.
10. No history of inflammatory rheumatism, definitely infected tonsils or other significant infections.
11. Examination either at home office or by a well qualified examiner.

On two occasions our mortality experience in this group has been presented to this Association. First, at the twenty-ninth annual meeting, held in January 1919 (see Vol. V., 1917-1918, pages 360-361); again at the thirty-eighth assembly in October 1927 (Vol. XIV, pages 173-180), which brought the results down to the anniversaries in 1924, including 2,669 lives.

This year a further study has been made by adding 1,126 new lives, insured during the three years 1924, 1925 and 1926 carried to the anniversaries in 1932. It is gratifying to find that the mortality is more favorable now than at the time of the 1927

Systolic Heart Murmurs

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The Northwestern Mutual Life Insurance Company
Heart Murmur Mortality—Risks Accepted—Issues 1905-1926
To Anniversary in 1932—American Men Table
(Select and Ultimate)

Under 40 Years of Age

| CLASS | NO. OF LIVES | 1ST 5 YEARS | | | AFTER 5 YEARS | | | ALL YEARS | | |
|-------|--------------|-------------|------|-------|---------------|------|-------|-----------|------|-------|
| | | Exp. | Act. | % | Exp. | Act. | % | Exp. | Act. | % |
| I | 1486 | 28.75 | 28 | 97.39 | 61.34 | 60 | 97.82 | 90.09 | 88 | 97.68 |
| II | 702 | 13.07 | 13 | 99.46 | 26.48 | 13 | 49.09 | 39.55 | 26 | 65.74 |
| III | 349 | 6.56 | 4 | 60.98 | 8.19 | 6 | 73.26 | 14.75 | 10 | 67.80 |
| IV | 135 | 2.58 | 2 | 77.52 | 3.34 | 0 | — | 5.92 | 2 | 33.78 |
| Total | 2672 | 50.96 | 47 | 92.23 | 99.35 | 79 | 79.52 | 150.31 | 126 | 83.83 |

Age 40 and Over

| CLASS | NO. OF LIVES | 1ST 5 YEARS | | | AFTER 5 YEARS | | | ALL YEARS | | |
|-------|--------------|-------------|------|--------|---------------|------|-------|-----------|------|-------|
| | | Exp. | Act. | % | Exp. | Act. | % | Exp. | Act. | % |
| I | 705 | 30.11 | 23 | 76.39 | 75.79 | 61 | 80.49 | 105.90 | 84 | 79.32 |
| II | 237 | 10.54 | 8 | 75.90 | 27.11 | 16 | 59.02 | 37.65 | 24 | 63.75 |
| III | 122 | 5.40 | 6 | 111.11 | 8.41 | 7 | 83.23 | 13.81 | 13 | 94.13 |
| IV | 59 | 3.07 | 0 | — | 4.21 | 2 | 47.51 | 7.28 | 2 | 27.47 |
| Total | 1123 | 49.12 | 37 | 75.33 | 115.52 | 86 | 74.38 | 164.64 | 123 | 74.71 |

All Ages

| CLASS | NO. OF LIVES | 1ST 5 YEARS | | | AFTER 5 YEARS | | | ALL YEARS | | |
|-------|--------------|-------------|------|-------|---------------|------|-------|-----------|------|-------|
| | | Exp. | Act. | % | Exp. | Act. | % | Exp. | Act. | % |
| I | 2191 | 58.86 | 51 | 86.65 | 137.13 | 121 | 88.24 | 195.99 | 172 | 87.76 |
| II | 939 | 23.61 | 21 | 88.95 | 53.59 | 29 | 54.11 | 77.20 | 50 | 64.77 |
| III | 471 | 11.96 | 10 | 83.61 | 16.60 | 13 | 78.31 | 28.56 | 23 | 80.53 |
| IV | 194 | 5.65 | 2 | 35.40 | 7.55 | 2 | 26.49 | 13.20 | 4 | 30.30 |
| Total | 3795 | 100.08 | 84 | 83.93 | 214.87 | 165 | 76.75 | 314.95 | 249 | 79.06 |

I—Murmur reported previously but not found at the time we assumed the risk.

II—Non-organic murmur found at time of examination.

III—Non-organic murmur found by Heart Specialist or at Home Office.

IV—Heart enlargement reported previously but not found at the time we assumed the risk.

The Northwestern Mutual Life Insurance Company
Deaths in Groups 1—2—3—4
of
Heart Murmur Mortality Classification

| CAUSES OF DEATH | GROUP 1 | | GROUP 2 | | GROUP 3 | | GROUP 4 | | TOTAL | |
|------------------------------|---------------------|---------------------------|---------------------|---------------------------|---------------------|---------------------------|---------------------|---------------------------|---------------------|---------------------------|
| | Ages under 40 | Ages 40 and over | Ages under 40 | Ages 40 and over | Ages under 40 | Ages 40 and over | Ages under 40 | Ages 40 and over | Ages under 40 | Ages 40 and over |
| Accident | 9 | 3 | 4 | 1 | 0 | 2 | 0 | 0 | 13 | 6 |
| Actinomycosis | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Alcoholism | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 0 |
| Anemia—pernicious | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| Aneurysm, aortic | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Angina Pectoris | 1 | 7 | 1 | 2 | 0 | 0 | 0 | 0 | 2 | 9 |
| Apoplexy | 1 | 6 | 0 | 1 | 1 | 0 | 0 | 0 | 2 | 7 |
| Appendicitis | 1 | 3 | 2 | 0 | 1 | 0 | 0 | 0 | 4 | 3 |
| Bladder Rupture | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Brain Tumor | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| Brain Softening | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Cancer | 5 | 13 | 2 | 6 | 0 | 3 | 0 | 0 | 7 | 22 |
| Coronary Thrombosis | 3 | 2 | 1 | 0 | 0 | 1 | 0 | 0 | 4 | 3 |
| Diarrhoea and Enteritis .. | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Diabetes | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 |
| Diphtheria | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| Disease of Nervous Sys... | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Disease of Arteries | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 |
| Encephalitis | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 2 |
| Endocarditis | 7 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 7 | 3 |
| Epilepsy | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Gangrene | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Heart Disease | 13 | 16 | 2 | 6 | 0 | 0 | 0 | 0 | 15 | 22 |
| Hodgkins Disease | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Intestinal Obstruction | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Leukemia, acute | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Liver Disease | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 2 | 2 |
| Nephritis | 2 | 6 | 1 | 3 | 1 | 0 | 0 | 0 | 4 | 9 |
| Paralysis, general | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 0 |
| Pneumonia—Influenza | 14 | 9 | 4 | 0 | 0 | 0 | 0 | 0 | 18 | 9 |
| Rheumatism | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 |
| Senility | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Septicemia | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| Skin Disease | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 0 |
| Stomach Ulcer | 0 | 2 | 2 | 0 | 0 | 1 | 0 | 0 | 2 | 3 |
| Sudden Death | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| Suicide | 8 | 5 | 1 | 0 | 0 | 2 | 0 | 0 | 9 | 7 |
| Syphilis | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Tuberculosis | 7 | 1 | 3 | 0 | 0 | 0 | 0 | 0 | 10 | 1 |
| Typhoid | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 0 |
| Unknown Cause | 1 | 2 | 1 | 0 | 1 | 0 | 1 | 2 | 4 | 4 |
| TOTAL | 88 | 84 | 26 | 24 | 10 | 13 | 2 | 2 | 126 | 123 |

report. It should be noted especially that the improvement has been relatively greater in the under age 40 group. Six years ago the tabulation of the causes of death showed that 15 died of pneumonia and influenza under age 40, 8 of which occurred during the first flu epidemic, and now the comparable number is 18, thus seemingly confirming the opinion that the flu deaths were the disturbing factor.

The average age at entry of those who died is:

| Under 40 | 40 and Over | All Ages |
|----------|-------------|----------|
| 31.36 | 48.36 | 39.76 |

and the average duration of their policies is:

| Under 40 | 40 and Over | All Ages |
|----------|-------------|----------|
| 8.63 | 9.42 | 9.02 |

DR. SCADDING—For several years we have been following the recommendations of the Joint Committee on Jumbo Risks regarding X-ray and electrocardiographic requirements in cases applying for large amounts of insurance. We have naturally all had our problems of interpretation, so the next paper will be of considerable interest to every one of us and will be presented by Dr. Harold E. B. Pardee of New York City. Dr. Pardee is an internist of wide reputation, a graduate of the College of Physicians and Surgeons, Columbia University, and of New York Hospital. He is Associate Attending Physician, New York Hospital; Assistant Professor of Clinical Medicine, Cornell Medical School; Associate Attending Physician in Cardiology at Polyclinic Hospital, and Consulting Physician in Cardiology at the Women's Hospital.

THE THEORY AND PRACTICAL APPLICATION OF THE ELECTROCARDIOGRAM IN LIFE INSURANCE

BY HAROLD E. B. PARDEE, M. D.

New York City.

The electrocardiogram is due to the electrical production associated with the chemical changes underlying the contraction of the heart muscle. Each muscle unit gives rise to a series of electrical potentials. First a quick diphasic movement and then a

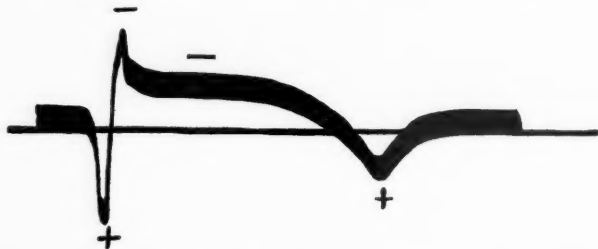


FIGURE 1—Deflection obtained by direct lead from cardiac muscle. (After Craib) *Reproduced from Clinical Aspects of the Electrocardiogram.* Harold E. B. Pardee; 3rd Edition; Hoeber Inc.; New York; 1933.

more prolonged slower movement which produces a peak in the same direction as the first deflection. A record of this might be called an electromyogram. (Figure 1.) The waves of the electrocardiogram are produced by the summation of the multitudes of these small units arising simultaneously in the various parts of the cardiac muscle which are active at a given time.

A record of the electrocardiogram may be obtained by placing electrodes in contact with any two portions of the surface of the body and leading the current through a galvanometer. Three standard leads are used for clinical work and these are Lead 1 from the right arm and left arm, Lead 2 from the right arm and left leg, Lead 3 from the left arm and left leg.

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By each of these leads we obtain a series of waves having a general similarity, yet different in their details. There is first a small rounded wave called P due to the activity of the auricles; next a series of sharply pointed waves—the QRS group which is due to the beginning of the activity of the ventricles, following which there is a relatively simple wave called T due to the final activity of the ventricles.

It may seem surprising that the electrocardiogram of one individual is so different by each of the three leads but this is due to the fact that the different pairs of extremities of which the three leads are composed are in relation to different portions of the heart. A reference to Figure 2 for instance will show that of the lead 1 pair, the right arm is in relation to the basal or

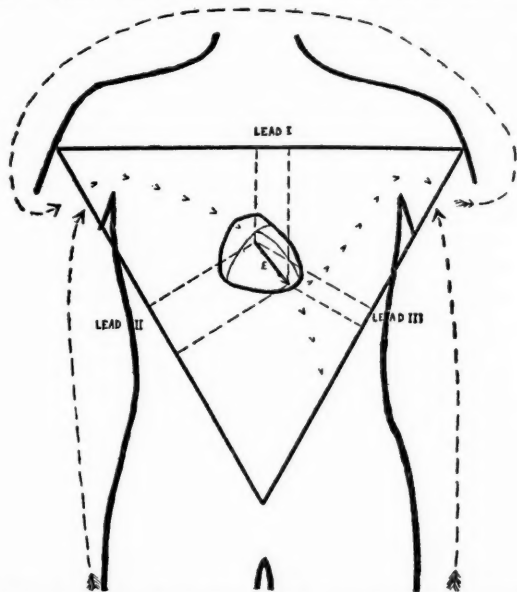


FIGURE 2—Illustrating the relation of the current within the heart to that obtained by the three leads. The body is viewed from the anterior aspect. Each side of the triangle between the right arm, the left arm and the region of the legs (left leg) represents one lead. *Reproduced from Clinical Aspects of the Electrocardiogram. Harold E. B. Pardee; 3rd Edition; Hoeber Inc.; New York, 1933.*

auricular portion of the heart and the left arm to the left border or ventricular portion, whereas in the lead 3 pair of extremities the left arm makes contact as above with the left border whereas the other contact through the left leg is in closest relation to the inferior portion of the heart which is composed of both right and left ventricles.

There has been some confusion in the terminology of these waves because Lewis did not follow Einthoven's original plan and because later writers have not unanimously followed either one. The majority, however, have followed Lewis' interpretation of Einthoven's terminology and this is the one which forms the basis of most of the recent studies. According to this terminology the auricular wave (P) may be spoken of as upward, downward, isoelectric (showing no deflection), notched or diphasic (having both an upward and a downward deflection). The QRS group is composed of two or three deflections as a rule. If there is an upward deflection this is called R. Any downward deflection preceding this is called Q, while a downward deflection following it is called S. If there is only a downward deflection though now usually called S this had better be called a Q. There may occasionally be more than one upward deflection and occasionally more than two downward ones. No satisfactory nomenclature has been suggested for the waves of such complexes but the term vibratory QRS has been applied to certain ones which show two or more upward and two or more downward peaks.

The T wave is also spoken of as being upward, downward, isoelectric or diphasic. Some confusion has resulted from the introduction of the term S-T interval or S-T segment which has been applied to the portion of the T wave immediately after the ending of the QRS group and before the peak of T. This portion of the curve is occasionally isoelectric, i.e., showing no deflection for a period of from .02 to .08 second or more. It is then, and then only, a true S-T interval. In other records this segment shows a definite deflection from the first, with a gradual transition to the peak of T. To speak of an S-T interval produces confusion for it seems to mark off a part of the

T wave from the remainder of this wave. This is a distinction which in some records is difficult or impossible to make. It seems better for the present to consider the whole of the electrocardiogram following QRS as the T wave. The portion of the curve which marks the beginning of T may be spoken of as the S-T transition or junction.

It might be well at this point to discuss what types of T wave should be called diphasic. There has not yet been general agreement on this point but to the author it seems best to speak of this wave as diphasic only when it has two definite apices, in opposite directions so that the movement of the string shadow is first in one direction and then in the other, thus forming a second peak, with a direction opposite to that of the first. This would exclude certain T waves which show the S-T transition on the opposite side of zero from the peak of T but which show a uniformly upward or downward movement of the deflection from the S-T transition to the peak. It would not exclude T waves with the S-T transition at a distance from zero curving first toward it and then away from it to form the peak.

Our knowledge of the clinical significance of the electrocardiogram has been obtained by studies of the variations of the waves obtained from normal hearts and of those observed in records from hearts affected by disease. Though there is a great variability in each feature of the electrocardiogram obtained from different normal hearts, yet certain types of variations do not occur in such records. These more unusual features we associate with an abnormal muscular activity.

The height, the duration and the form of any or all of these waves will be modified by factors which change the character of the spreading of the contraction over the muscle of the auricles or of the ventricles, or which interfere with the contraction itself. Such factors include certain drugs and toxins and certain disease processes such as hypertrophy, inflammation or degeneration. There are likewise metabolic disturbances of the muscle, such for example as those produced by hyperthyroidism or marked anemia, which may affect the form of these waves. Because the record may be affected in so many ways by so many

factors, electrocardiographic diagnosis demands a knowledge of the normal variations of the waves and the recognition that abnormal features may result from the action upon the myocardium of drugs, toxins, metabolic disturbances or disease.

It is necessary to study these electrocardiograms very carefully in order to group them into similar categories and the basis for this study must be measurement, combined with a consideration of variations in the form of the waves. There should be measurement of the duration of the waves, of the intervals between them, and of the height of the waves, also a notation of the upward or downward direction of each wave in the three leads of the record and a description of any special feature of the form of a wave which may be present.

In measuring duration it is important to realize that the duration in one lead may not be the same as the duration in another. As has been said, one lead may be so situated in relation to the current within the heart producing a wave that there will be no deflection in that lead. Such a wave we speak of as being absent or isoelectric in this lead but in such a case the other two leads will always show the deflection. If only a portion of a wave is isoelectric in one lead there will be a shortening of the duration of this wave in that lead as compared with its duration in the other leads which record this isoelectric portion. It is for this reason that measurements of duration must always be made in three leads and the longest measurement selected as being the nearest approximation to the duration of the electrical disturbance in the heart.

The measurement of the duration of the P wave indicates the duration of the spreading of the contraction throughout the auricles and does not normally exceed .10 second. The measurement of the P-R interval is made from the beginning of P to the beginning of QRS and indicates the time elapsing from the onset of auricular activity to the onset of ventricular activity. This gives a measurement of the time taken for the spreading of the contraction across the auricles and through the auriculo-ventricular bundle and its ramifications to the ventricular muscle and should not exceed 0.18 or 0.20 second with normal heart

rates. The duration of the QRS group indicates the time taken for the contraction to spread throughout the ventricular muscle and should not exceed .10 second. At the end of QRS the spreading of the contraction is finished and the duration of T indicates the time during which there continues to be an electrical production from ventricular activity. Its normal duration depends in part upon the heart rate, being found shorter with more rapid rates but this measurement has not yet yielded any clinically useful information.

In measuring the height of the waves it is necessary to determine the isoelectric or zero level of the record. This must be taken as that portion of the record immediately preceding the P wave unless this region should be occupied by a small elevation called U which follows the preceding T, or, as is common with heart rates over 100, by the T wave of the preceding cycle. In order to properly measure the height of the waves the isoelectric level must be parallel to the horizontal line of the record. In records which fail to show the zero level of the record it might be approximated as the level immediately following the P wave, but the statements in the following paragraph should be considered in this connection.

A special precaution is necessary in measuring the first portion of the T wave; the S-T transition. Following P, between it and QRS, there is usually a slight deflection of less than 1 mm., in a direction opposite to that of the peak of P. It is for this reason that the level between P and QRS is usually different from that preceding P. This deflection is due to a continuation of the auricular activity and constitutes the final or T deflection of the auricular contraction. In records showing complete heart block it may be observed to last until about .36 second after the beginning of P so that it will influence any ventricular deflections which occur during this time. It would seem proper then to use the P-R level as the zero from which to measure the deflections of QRS and of the first portion of T especially the S-T transition. This dislocation of the zero level is usually so slight that it is of little importance in relation to the large waves of the QRS group but in relation to the S-T transition it may be of considerable importance.

One object in measuring the height of the different waves in each of the three leads is to determine the largest deflection for each of the three chief divisions of the electrocardiogram, i.e., P, the QRS group and T. This affords an approximation of the voltage within the heart giving rise to these waves and is usually found in only one of the three leads. It may be either an upward or a downward deflection but we are concerned only with its absolute value in millimeters above or below zero, for this expresses its magnitude. The voltage of P should exceed 1 mm. The voltage of the QRS group should exceed 5 mm., and the voltage of the T wave should exceed 1 mm. The S-T transition should not be deflected as much as 1 mm. from the zero indicated by the level between P and the QRS group.

There is one portion of the QRS group whose size has a special importance, namely, the Q wave of lead 3. Q-3 is an abnormal finding only if in the absence of right axis deviation of QRS it reaches a size which equals or exceeds 25 per cent. of the voltage of QRS. It must be emphasized that a downward deflection which is preceded by an upward one is not to be called Q no matter how small the initial upward deflection may be. The objection to naming such waves Q is that it would be necessary to define how small the initial upward deflection must be in order to be disregarded. This definition has not yet been written and would be extremely difficult or impossible to devise.

After measurement we must consider the directions of P, QRS and T in the three leads noting whether the deflection is upward or downward in each lead. Both P and T should be found upright in leads 1 and 2. T should not be isoelectric or diphasic in lead 2 but should show a definite upward deflection. The chief deflection of QRS is usually upward in all three leads of normal records but is occasionally downward in lead 1, or lead 3. When there is a definite S or Q in lead 1 and the height of R-2 is less than the height of R-3 the condition is called right axis deviation of QRS and is often though not always due to right ventricular preponderance because of hypertrophy. When there is a large Q or S in lead 3 and R-2 is less than R-1 this con-

dition is called left axis deviation of QRS and this is often though not always due to left ventricular preponderance because of hypertrophy.

An abnormal feature of the QRS group deserving special mention is the occurrence of notching or slurring of the waves either at the peak or on the upward or downward limb. It should be emphasized that this notching or slurring must be distinct and must occur either at the peak or in the upper half of a wave which represents the principal deflection of the QRS group and that some sign of this notching or slurring must be discernible in two leads. Slurring and even notching without these qualifications may be found in records from hearts which are in no way abnormal. With these qualifications a positive finding will probably always indicate a significant disturbance of the spreading of the contraction wave which in its turn is due to myocardial disease.

Beside actual inversion of T in lead 1 or 2 there are certain deformities of this wave which have an abnormal significance. It should not be found diphasic in either of these leads. Moreover the character of the curve from the S-T transition to the peak of T is important. It should be a smooth curve, concave

TABLE 1

P Wave

Low voltage (less than 0.7 mm.)
High voltage (more than 2.0 mm.)
Diphasic in Lead 1 or 2
Inverted in Lead 1 or 2
Abnormal duration (0.12 sec. or more)

QRS Group

Low voltage (5 mm. or less)
High voltage (more than 17 mm.)
Abnormal duration (0.12 sec. or more)
Notched (as described in text)
Large Q-3 (as described in text)

T Wave

Low voltage (1 mm. or less)
High voltage (more than 5.5 mm.)
Diphasic in Lead 1 or 2
Inverted in Lead 1 or 2
S-T transition (1 mm. or more from zero)
Abnormal form of curve to peak of T

in the direction of the peak of T. The apex of the peak in leads 1 and 2 should have a characteristic, slightly rounded, somewhat pointed form. If the line from the S-T transition to the peak of T does not show this concavity or if the peak of the wave is blunt or flattened or doubled it is an abnormal feature. Table 1 will serve as a summary of these abnormalities and if one of these is found it is an indication that something is abnormal about the contraction of the portion of the heart which produces the abnormal wave.

In addition to the abnormalities of the form of the electrocardiogram there are three abnormalities of rhythm which have a quite definite pathological significance. These are auricular fibrillation, auricular flutter and ventricular tachycardia. These conditions may occasionally occur transiently in normal individuals subject to certain toxic influences but with this exception can probably always be taken as an indication of myocardial disease.

The appearance of premature beats or paroxysmal tachycardia in a person of forty years or more even without other signs of heart disease, is a suspicious feature unless this person has been known to have had them since early life. When these arrhythmias first appear in the arteriosclerotic age they are often, though not always, an indication that an area of the myocardium is failing to receive a proper blood supply owing to narrowed arterial branches.

Having measured and examined these waves with care, we shall be in a position to say whether we are dealing with a normal or an abnormal electrocardiogram. From the insurance point of view you will wish to know what significance is to be attached to this conclusion. You must remember that a normal electrocardiogram indicates a normal contraction process in a normal myocardium. It does not indicate the condition of the valves or of the pericardium and up to a certain point it does not indicate the condition of the coronary arteries. If the valves or the pericardium or the coronary arteries are diseased and if the disease in these portions of the heart has affected the structure or the function of the myocardium, the electrocardiogram will then register this fact by showing some abnormal

feature. If these conditions have not affected the structure or function of the myocardium the electrocardiogram will be normal.

There is still another combination of circumstances which may afford an apparently normal electrocardiogram in spite of the presence of myocardial abnormality. A record having a normal appearance may yet, if compared with a previous record from the same individual show so much difference as to indicate that some toxin or disease must have affected the heart to produce the change. For this reason, previous electrocardiographic records are of value in making a decision as to whether or not the myocardium is normal. A series of records unchanged over a period of years should be very reassuring in deciding to accept cases who return for larger amounts of insurance.

It must be remembered that moderate degrees of right or left axis deviation of QRS are occasionally found in records from apparently normal individuals and in such cases are probably due to cardiac structural peculiarities of a congenital nature which have no determining effect upon the function of the heart or the longevity of the patient. On the other hand the finding of this peculiarity should always lead to a search for some condition which might lead to hypertrophy of the ventricle indicated.

We must especially remember that in normal individuals with the hypersthenic habitus the high position of the diaphragm may raise the apex of the heart and rotate it upward and toward the left giving rise to left axis deviation of QRS. In such cases there is also likely to be an isoelectric or downward T wave in lead 3. Obesity has a similar effect upon QRS and T, tending to produce left axis deviation of QRS and a downward T-3; even P-3 is sometimes inverted in records from obese patients. There is of course no special cardiac implication connected with such findings in an obese individual.

It must be borne in mind that a large Q-3 is an occasional finding in records from normal hearts during pregnancy and that it may occur very rarely—less than 1% in records from supposedly normal males. As far as we know these records are only obtained from individuals with obesity or a high level of

the diaphragm, both of which cause the heart to lie more transversely than normal within the thorax.

Prolongation of the P-R interval may sometimes be due to vagotonia and in such cases will be abolished by atropine in large doses. If not abolished by atropine this defect in the conduction of the stimulus from the auricles to the ventricles should probably be taken as an indication of disease of the auriculoventricular bundle. The possibility of its being due in rare cases to a congenital peculiarity has not yet been definitely proven but the following observations lend some support to this idea. In Lewis and Gilder's series of 52 normal individuals only one was found with a P-R interval greater than 0.2 second, but in a series of 1,800 normal individuals Ferguson and O'Connell found 26 records with the P-R interval exceeding this figure. Eighteen of these records showed the P-R interval constantly above the normal figure, while in the other eight the measurement varied from time to time between a normal figure and one greater than normal. Completely blocked auriculoventricular impulses never appeared in these records though P-R measured as much as 0.29 in some cases. Four of those with constantly prolonged P-R were tested with atropine sulphate grain 1/30th hypodermically. In one case the measurement was reduced from 0.22 second to 0.18 second; in one from 0.23 second to 0.20 second; in one from 0.24 second to 0.22 second, while in the other the measurement remained at 0.29 second after atropine as it had been before.

Having found an abnormal electrocardiographic feature how should this be appraised? It is important from the life insurance point of view to accumulate facts upon the relation of peculiarities of the electrocardiogram to variations in longevity. In order to do this it will be necessary as Dr. Fellows has previously pointed out, to collect cases with similar electrocardiographic features and to follow the patients over a period of years to determine the duration of their life. The medical practitioner sees electrocardiograms which are obtained almost exclusively from individuals who have more or less definite cardiac complaints. It is not proper to flatly apply the prognosis

obtained from such material to the type of person who would present himself for life insurance. Proper knowledge can only be acquired from follow-up studies of patients showing these abnormalities, but pending the completion of such studies some procedure must be adopted. The general life experience of the past has been accumulated without the aid of electrocardiographic findings. The use of data other than the electrocardiogram should be able to give as good results as have been obtained in the past. There is danger of attaching too much influence to a peculiarity of the record and thus rejecting a proper risk. On the other hand it is evident, even at the present state of our knowledge, that the records may be helpful in detecting otherwise hidden disease. A person otherwise perfectly negative but with one of the abnormalities of the ventricular complex shown in the table should certainly be considered as having an exceptional heart and with proper exclusion of toxic influences in occasional cases there is a practical certainty that such a heart is affected by disease. Abnormalities of the auricular waves are not perhaps so definitely incriminating in the present state of our knowledge as abnormalities of the ventricular waves. They should nevertheless, be given due weight as indicating a state of abnormality of the auricular muscle contraction.

The actual prognosis of a patient showing an electrocardiographic abnormality does not depend upon this finding alone. It depends upon the etiology of the disease which has produced the change in the myocardium; it depends upon the tendency of this causative disease to respond to treatment; it depends upon the type of life to which the patient will subject his abnormal cardiac mechanism.

These variables will divide up the patients showing a given abnormality of the electrocardiogram into so many groups that a tremendous number of observations will be necessary to determine the special significance of each category. The value of the electrocardiogram does not lie so much in its indication of the degree of cardiac impairment as in its ability to help in discovering those patients who have a structural abnormality of the heart muscle itself, or some functional abnormality of the muscle

due to disease elsewhere in the body. When an abnormal electrocardiogram is the sole abnormal finding I am convinced that better mortality figures will be obtained in a group without these abnormalities, just as better mortality figures are obtained in a group without murmurs at the valve areas of the heart. On the other hand it is possible to sort over those patients with electrocardiographic abnormalities as well as those with murmurs at the valve areas of the heart and select from each group especially favorable cases in whom the mortality will not differ greatly, if at all from the normal. The basis on which this selection should be made for patients with abnormal electrocardiograms must be determined by the follow-up of rejected risks but it seems at present that transient arrhythmia, prolongation of the P-R interval and the right or left axis deviation of QRS should be considered less seriously than the abnormalities of the ventricular complex listed in the table or even than abnormalities of P.

DR. BIRCHARD—This privilege of discussing Dr. Pardee's paper is both easy and difficult. He has performed an extraordinary compression of a large amount of material and information, and for that reason it is rather easy and also for the reason that he has done it awfully well. It is difficult because I can't find anything to criticise. There are one or two things about which I would like to have had a disagreement—pet hobbies of my own—but I find I cannot do it. Anyway, it is far above my ability to criticise anything that Dr. Pardee says about electrocardiograms. Since the original masters, Doctors Einthoven and Lewis, there has been no person who has done so much for the art and science of electrocardiography as has Dr. Pardee. If you read the electrocardiographic literature of the times, you will find Pardee constantly quoted, very frequent articles from his pen, and then there is his very well known book on the subject, which is extremely readable and which you will find very valuable to possess and peruse.

There is one thing I would like to have seen Dr. Pardee deal with to some extent, and which he has been undoubtedly re-

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strained from doing by lack of time, and that is to go back to the primary fundamentals of the electrocardiogram. Had he had the time he could have given us, particularly those with any flare for physics (and physics is quite the king of the sciences at the present, mathematics still being regarded as the queen), he could have taken you inside the individual muscle cell and shown you a beautifully equipped physical laboratory that would have been very, very interesting indeed. He could have made you visualize the movement of the electrons across the cell membranes that would have given you a personal interest in electrocardiograms which you otherwise are not likely to have. I do not know any phase of medicine in which you get any closer to the actual fundamentals of life than you do in the electrocardiogram. Some time in the not very distant future, the physicists are going to give a clearer conception of what goes on in these muscle cells, including why a heart cell ceases to function; and if we learn that, we will probably know why a heart cell ever does function. When that time arrives, the cardiologist will have something more to his armamentarium than digitalis, common sense and rest, which is about all he has at the present time.

You will have noticed that Dr. Pardee has agreed that the nomenclature of the waves that constitute the electrocardiogram have not been too well named. This is quickly apparent if you try to analyze electrocardiograms carefully and intelligently; and, by the way, you mustn't attempt to analyze electrocardiograms unless you go back to the fundamentals of the thing, but it is very easy if you do go back to the fundamentals. Any of you can read them pretty successfully if you put one month of work on the thing and start at the beginning and come up consecutively. The names of the waves are undoubtedly badly chosen. The "Q" wave in Lead I may not be a "Q" wave in III, etc. I have adopted, for personal use (I don't use it in reports to physicians) a scheme whereby the QRS deflections are styled "ventricular event one", "ventricular event two" and "ventricular event three", etc., and I try to identify, we will say, "ventricular event two" of Lead I with "ventricular event two" of Lead II and Lead III. One can do it easily enough with a

little study. I think that sometime an alternative and more clearly understandable nomenclature can be developed.

I expected Dr. Pardee would tell you that the left axis deviation doesn't amount to very much. I observe he is of the opinion that left axis deviation, that condition of QRS deflection group which indicates that the voltage developed by the heart instead of running downward and to the left from the center of the chest runs upwards toward the left shoulder, may occur in normal individuals, and that it is really more or less of an accident which does not amount to much and may be due to a high diaphragm in an overweight person. I am firmly convinced that we, as insurance people, will eventually be able to prove that it does mean very considerable in terms of mortality. At the same time I admit that it may be due to a high diaphragm in an overweight person. I was interested in some data that Dr. Fellows and Dr. Turner handed around to some of us about a year and a half ago with respect to the work they had done in taking about thirteen hundred electrocardiograms which they had divided into cardiovascular normals and abnormals. As a bit of mathematical amusement one day, after making certain assumptions as to ages, I ran out the probable errors of the percentage incidence of left axis deviation in the two groups. The probable error of the difference of the percentage indicated that left axis deviation is of clinical interest. The chances appeared to be about nineteen out of twenty that left axis deviation is significant of a pathological process of some sort. Right axis deviation in a person who has not a congenital heart disease or who is not suffering from mitral stenosis, you will see occasionally in life insurance and occasionally in hospital practice. I have been very much concerned as to what those actually mean. Those who come to the Sun Life, we decline outright and assume that something or other has happened in the ventricle to disturb the ordinary and proper spread of the excitation wave over the ventricle and that something we think has been an arterial catastrophe of some kind, even in the absence of a positive history.

There is one other point I would like to mention: that is with regard to electrocardiograms obtained for life insurance purposes.

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You have all seen the little booklet gotten out and handed around to medical directors and examiners, which suggested a film rate of travel in making electrocardiogram of from 35 to 40 millimeters a second. The electrocardiograms so obtained are much more readable than with the present speed of 25 millimeters per second. The idea was that the splintering and the "slurring" of the QRS deflection would be more manifest and not overlooked so often, and that we would eventually get some statistical information as to what those things mean in terms of mortality.

Having done a considerable amount of electrocardiography for nineteen years and having also been connected with a life insurance company for a major portion of that time, I am rather inclined to be a little more rigorous in my demands as to what constitutes normality of the electrocardiogram than is the average pure clinician. This applies to all of us who have had much training in life insurance. We regard things as abnormal that the man who is a pure clinician does not regard as abnormal. For that reason, my own demands as to what constitute an abnormal electrocardiogram are fairly rigorous. On the other hand, one constantly sees electrocardiograms handed around where medical consultants and medical directors have taken too rigorously severe positions. Every week sees such electrocardiograms come from other life offices which I don't think amount to anything at all and will have no effect on mortality. In those offices where you send out your electrocardiograms to a clinician for interpretation, there appears to be great lack of uniformity of interpretation. I know one very well known clinician, well known to a lot of you people, and his interpretations of electrocardiograms for various life insurance companies are so extraordinarily lenient that I think it would be unwise to follow them. On the other hand, I know of a few clinicians who are very, very much more rigorous than I would think of being.

It was a very great treat to see Dr. Pardee, in his very skillful way, compress a large volume of material into a very small compass and present it in a way that all of us, whether we have had any experience in electrocardiograms or not, can understand easily and thoroughly.

DR. TAYLOR—As Dr. Pardee brings out, the statistics to date have been chiefly gathered from the study of cases where cardiovascular pathology existed or was suspected. Insufficient evidence has been gathered as to the significance of certain electrocardiographic changes in supposedly normal individuals. From the medico-actuarial viewpoint, such insurance statistics as exist are lacking both in volume and time-spread to be of any definite value. Obviously the answer is careful records of such cases by all companies over a period of years. These records should be so cross-indexed that with a minimum of effort a study of a given impairment may be made. After a few years the volume of evidence so gathered will be such that some conclusions can be drawn; but in the meantime, how is the medical underwriter to act on these cases?

Seeing the need of concerted action on the subject, Dr. Christiernin, in April, 1932, called together a group of members who were especially interested in the subject to formulate a tentative schedule for our guidance. Naturally there was some divergence of opinion on various features of the subject, but after several conferences, such a schedule was drawn up and subscribed to by all the committee. In some respects this schedule is probably very liberal (e.g.: P-R and QRS time interval limits); in others possibly too stringent (e.g.: low voltage QRS in absence of other changes); and we may find that certain changes which now call for rejection may be taken by careful selection on substandard basis.

A report of this early work of the committee was given at our last meeting, and the group which had informally discussed the subject was made a permanent committee to continue with this study. With the exception of myself—and this is not a modest gesture on my part—the members of this committee are eminently fitted by training to guide us in our handling of X-ray and electrocardiographic matters. If the individual companies will cooperate by keeping usable records and make occasional "follow-ups" as requested, we will in time gather a volume of evidence that will be impressive and of benefit, not only to us as underwriters, but to the clinicians as well.

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But to get back to the discussion—I am glad that Dr. Pardee reminds us that we still have available all the old procedures for cardiac diagnosis. We are too prone to let a machine substitute for the five senses and it is a mistake to allow various electrocardiographic changes, the significance of which is not understood, to so confuse our judgment that we lose sight of satisfactory clinical evidence. On the other hand, a normal electrocardiogram means simply normal conduction and tells us nothing as to the condition of the endo or pericardium.

However, there are certain changes in the ventricular portion of the electrocardiogram which label such a case uninsurable regardless of clinical findings, such as:

- (a) Prolongation of QRS interval over .1 sec.
- (b) Apex notching of the QRS group in all leads.
- (c) Iso-electric, diphasic, or negative T waves in Leads I or II.
- (d) Low voltage T waves in all leads.
- (e) Coronary T waves in any lead.

All of these changes are indicative of myocardial pathology and may not be detected by any other means. A case in point:

Man—aged 47—applied for total of about one million in eight companies—we were offered 125 M. Past history, clear. Physical 1-10-31—showed pulse 92. B.P. 154-86 ascribed to nervous tension incident to examination and the fact that two days previously he had had a dental extraction under novocaine. Two outstanding examiners of Chicago said he was a good risk. An E.K.G. of 1-23-31 showed T changes in Lds. I and II—none of the insurance applied for was delivered. Man died suddenly 5-10-31 (four months later) after a round of golf.

Abnormalities in the auricular portion of the electrocardiogram seem to be less significant as indicating impaired longevity. This is as we would expect, for a balanced circulation depends on an efficient left ventricle.

Further study is necessary before we can say we will disregard P changes that occur alone. Various degrees of heart block, of course, call for rejection, the outside limit of the P-R interval

being generally accepted as .2 sec. You will note the committee recommends rejection of cases where the P-R interval reached .22 sec. or the QRS interval .11 sec. They were agreed that the normal upper limits were .2 sec. and .1 sec., respectively, but to allow for the individual equation in measuring, they put down .22 and .11 as points at which they all agreed to decline.

Personally, I still question all cases where the QRS interval exceeds .08 sec. as we reported two years ago—in 22 cases showing a measurement between .08 and .1 sec. there were 9 deaths within a five year period and of the 13 living only one case was entirely clear, the others showing either T changes or cardiovascular changes.

The significance of a deep "Q" wave in Lead III has caused much comment pro and con. Dr. Pardee was the first to draw attention to it and he precipitated a discussion that is still going on. You will note that the committee sort of "straddle" on this point and put Q3 cases into the questionable group, allowing the individual underwriter to act as he sees fit. The majority of the committee, however, recommend rejection of such cases. Personally, I am following the crowd, the bulk of the evidence submitted to date seems to support Dr. Pardee's contention and until further evidence to the contrary is in, I feel we should stay off this class of risk.

I was interested in Dr. Pardee's reaction as to the significance of premature contractions in the older age group. I was taught as a student that premature contractions of themselves were of no significance as indicating pathology, regardless of age; but more detailed study since the arrival of the electrocardiogram has caused some doubt as to whether this held true for the older ages. The idea of viewing those cases differently where premature contractions had their onset after the age of 40 is new to me, but it seems plausible that such an occurrence might be evidence of sclerotic changes in the myocardium.

Much can be learned about a heart by studying successive electrocardiograms over a period of years. I believe it was Dr. Pardee in a former paper who likened an electrocardiogram to one's fingerprints, that successive tracings should be essentially

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identical, if no changes were taking place in the myocardium. When calling for an electrocardiogram also ask for photostats of any former tracings.

In closing let me again emphasize—there is a lot of work to be done on this subject and we can all help by keeping usable records.

DR. SCADDING—Dr. Pardee, have you some further comment to make?

DR. PARDEE—The only thing I want to say in addition to what I have already said is that it certainly would be helpful to take electrocardiograms at a somewhat faster speed than the 25 millimeters per second which is now standard. It helps in the measurement of the QRS interval and also in an observation of slurring and notching of QRS. It is advisable to get together on what you are going to call "notching" of the QRS group. There is the greatest chance there for individual impressions.

In regard to right and left axis deviation, I must say a word there. I think it is very important that you recognize that the records taken of young adults in the ages between eighteen and twenty-five definitely do show a certain percentage of right and left axis deviation records. The percentage is so large that I do not see how it can be accounted for by disease. Take the same individual at fifty-five years of age and he still has the right or left axis deviation. I believe that at fifty-five you are going to have much more than at twenty-five because time and wear and tear contribute to its increase.

DR. SCADDING—Our next paper will be presented by Dr. Henry B. Turner of the Metropolitan Life, in collaboration with Dr. Charles F. Nichols of the Penn Mutual and Dr. Harry E. Ungerleider of the Equitable Life Assurance Society. In a subject of such far reaching importance, I feel that we are particularly fortunate in obtaining the medical viewpoint of three of our larger life insurance companies.

A RECOMMENDED STANDARD FOR THE
DETERMINATION OF CARDIAC
ENLARGEMENT

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PART I—THE NORMAL TRANSVERSE CARDIAC DIAMETER

All observers who have worked extensively in cardiac Roentgenology agree that the orthodiagraphic method is more accurate by far than a simple postero-anterior film, but that when it is necessary to base decisions upon such X-ray plates alone, the transverse cardiac diameter¹ is the sole measurement which can be made with accuracy and with a simple correction for distortion as Hodges and others have shown, it falls within the normal limits as determined by the orthodiagram. The transverse diameter is influenced by enlargement of the right auricle and both ventricles. Information concerning the left auricle may be obtained in the oblique and lateral positions under the fluoroscope.

In insurance work it would be highly desirable to have a table offering definite figures of heart size for individuals of different habitus, based on the variables, height, weight and age, which can be accurately determined and will readily permit the collection of statistics in terms of percentages or actual variation above or below the predicted normal.

In 1931 the Heart Committee of the New York Tuberculosis and Health Association recommended the tables of Hodges and Eyster predicting cardiac area and cardiac transverse diameter

1. The transverse cardiac diameter is commonly obtained by adding the greatest distance of the right border of the heart to the greatest distance of the left border from the midsternal line.

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in the frontal plane from height, weight and age as criteria for determining the presence or absence of cardiac enlargement, the assumption being that these standards were more suitable than any other.

To check the accuracy of the predicted transverse diameter, 890 orthodiagrams of normal people in the erect or sitting erect position were collected at random from the literature wherever the age, height, weight and sex were given, and the total was brought to 1,000 from my own files.

Bainton in 1932 found that the prediction tables which were originally based upon males were applicable to females, provided a deduction of 8 mm. was made. This revised standard has been applied to the graphs of females, which in this series numbered 129.

The observations covered the ages from 15 to 63, weights from 100 to 216 pounds, and heights from 5 feet to 6 feet, 3½ inches. Analysis shows that approximately 8½% have transverse cardiac diameters which are more than 10% below the predicted value, that 95.8% do not exceed the predicted normal by more than 10%, that 3.1% lie between +10% and +12%, and 1.1% are greater than +12%, but less than +20%. From this it appears, as other observers have confirmed, that $\pm 10\%$ of the predicted value is a fair range for the normal heart, and that any heart exceeding this figure may be viewed with suspicion.

PART II—ABILITY OF THE PREDICTION TABLES TO SEPARATE NORMAL FROM PATHOLOGICAL HEARTS

Since the value of any method rests in its ability to segregate normal from definitely pathological hearts, Eyster in 1927 compared a series of 100 normal males with 125 patients. These latter included cases of mitral stenosis, mitral stenosis and regurgitation, aortic regurgitation, multiple valvulitis, and myocarditis. In these two groups, 3% of the normals exceeded +10%, whereas the weighted average of the pathological group showed 78% exceeding the normal by more than +10%.

In 1928 a second series composed of 100 normals and 100 cases of cardiac pathology without decompensation suffering from

diseases known to produce hypertrophy, namely mitral stenosis and regurgitation, aortic regurgitation, combined aortic and mitral disease, hypertension, and adherent pericardium, were examined. In this group again 78% of the pathological hearts exceeded the predicted normal by more than 10%.

Combining these researches, the figures show that in this group 93% of the hearts exceeding the normal by more than +10% belonged to those individuals with known cardiac pathology. Of the normals only 5.5% exceeded the predicted values by more than +10%. It is of interest that 149 cases showing only accidental murmurs, extrasystoles, or neuro-circulatory asthenia gave normal measurements.

PART III—COMPARATIVE MERITS OF VARIOUS CRITERIA

The final value of a particular standard depends not only upon its ability to segregate the normal from the diseased, but also upon its ability to do so more accurately than any other criteria in common use. The data in the second series of 100 normals and 100 abnormals were examined by various methods, including the commonly employed cardio-thoracic ratio which obtains the quotient of the transverse cardiac diameter divided by the maximum transverse internal thoracic diameter, which for years has been the common standard employed. Bainton in analyzing his 175 normals concluded that this ratio was incorrect and not applicable to orthodiagrams and further felt it was even less applicable to X-ray plates which contained even greater factors of error. Nichols in his review of heart size suggested that the cardio-thoracic ratio failed to detect approximately 30% of hypertrophied hearts. Bordet in France, who has had tremendous experience with X-rays as applied to the cardiovascular system, stated in 1931 that this criterion was too subject to error to be of value. In 1932 the Heart Committee of the New York Tuberculosis and Health Association concluded that "the so-called cardio-thoracic ratio is no reliable index of cardiac enlargement". It is a matter of note that most investigators who have worked extensively in the field have resorted to individual measurements based on the build of the individual rather than the simple ratios.

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Eyster applied eleven different criteria to the measurements of the normal and pathological cases and graded them according to their ability to segregate the two. The following is the order of value:

1. Cardiac area in the frontal plane, prediction based on age, height, weight. (Tables of Hodges and Eyster.)
2. The same as 1, with the addition of thoracic measurements.
3. Ratio of thoracic girth (measured at the 3rd intercostal space) to the cardiac area in the frontal plane.
4. Transverse cardiac diameter in the frontal plane, prediction based on age, height, weight and thoracic measurements.
5. Transverse cardiac diameter in the frontal plane, prediction based on age, height and weight. (Tables of Hodges and Eyster.)
6. Ratio of girth of thorax to transverse diameter of heart.
7. Sum of the transverse cardiac diameters in the frontal and lateral planes, prediction based on age, height and weight.
8. The same as 7, with the addition of thoracic measurements.
9. Area of frontal plane based on average, no prediction.
10. Cardio-thoracic ratio.
11. Transverse cardiac diameter in the frontal plane, no prediction, based on average.

Between the transverse cardiac diameter based on the prediction tables, and the same corrected for thoracic measurements, there is so little difference that it is not advisable to introduce the additional personal factor of obtaining the thoracic measurements.

From this critical study it appears that the most satisfactory criteria of heart size are based, first, on the predicted cardiac area in the frontal plane, secondly, on the predicted transverse cardiac diameter to which the addition of thoracic measurement offers a slight but impractical improvement, and that the cardio-thoracic ratio as ordinarily obtained is the least valuable with the single exception of the transverse cardiac diameter based on averages. Actually, only 42% of the pathological cases equalled or exceeded Danzer's upper limit of the cardio-thoracic ratio, whereas 78% of the pathological cases exceeded Eyster's predicted transverse diameter by more than 10%.

PART IV—SIX-FOOT PLATES—THE RELATIVE SUPERIORITY
OF THE PREDICTION TABLES OVER THE
CARDIO-THORACIC RATIO

Finally, although Hodges has shown clearly that the transverse cardiac diameter measured in X-ray plates taken at a distance of nine feet obeys the prediction tables after a slight correction for distortion has been made, it remains to show, first, that this standard is applicable to plates taken at a distance of six feet (the common practice in insurance work) where the distortion of the cardiac silhouette is increased due to greater divergence of the X-rays and where there usually is a disproportionate enlargement of the size of the chest and the size of the heart and, secondly, that the tables still remain a more satisfactory criterion than the cardio-thoracic ratio.

It is possible, by employing the average distance of the mean plane of the heart from the anterior chest wall and by knowing the distance from tube to film, to calculate the average increase which must be subtracted from the observed measurement to obtain a more exact estimate of the true transverse cardiac diameter or, alternatively, it is possible to obtain the exact distortion for a particular build by subtracting the orthodiagraphic transverse cardiac diameter from that found on X-ray plates.

With these facts in mind, three series of six-foot plates were examined and properly corrected.

1. Two hundred cardio-vascular normals were measured and found to conform to the prediction tables.
2. The first one hundred cases of persistent hypertension (elevation of both the diastolic and systolic pressures above 90 and 150 mm. of Hg., respectively, reported over a period of two years or more) were selected from the files. These were cases in which one might reasonably expect from the nature of the disorder to find a certain number with hypertrophied hearts. Further, only those cases were selected in which the cardio-thoracic ratio was normal. Of this group 22% showed varying degrees of enlargement, the transverse cardiac diameter exceeding the upper limit of normal as determined by the tables of Hodges and Eyster.
3. An additional series of plates was examined, composed of the first fifty cases of hypertension in which the cardio-

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thoracic ratio demonstrated cardiac enlargement. In no instance did the prediction tables fail likewise to show this condition.

Consequently, it appears that the prediction tables may be satisfactorily used with X-rays taken at a distance of six feet and are again more valuable than the cardio-thoracic ratio.

IN SUMMARY

1. Analysis of 1,000 orthodiagrams indicates that $\pm 10\%$ of the transverse cardiac diameter as predicted by the tables of Hodges and Eyster represents a fair range of the normal heart (this is in agreement with Eyster's original conclusion), that only 4.2% exceed the upper limit, whereas 98.9% do not exceed $+12\%$.

2. A deduction of 8 mm. from the prediction tables seems a fair standard for women.

3. A comparison of 200 normal individuals with 200 patients suffering from cardio-vascular abnormalities that produce hypertrophy shows that 93% of the hearts in which the transverse diameter exceeded $+10\%$ were definitely pathological.

4. In an analysis of postero-anterior films taken at a distance of six feet (the present practice in insurance):

- a. The transverse cardiac diameters of 200 individuals without cardio-vascular disease conform to the prediction tables of Hodges and Eyster.
- b. In a series of 150 persistent hypertensives the tables are more sensitive in detecting cardiac enlargement than is the cardio-thoracic ratio.

5. Application of various criteria to the same data shows that the transverse cardiac diameter based on the prediction tables of Hodges and Eyster is the most accurate standard available when an opinion is based upon postero-anterior X-ray plates, and that the cardio-thoracic ratio has relatively little merit.

RECOMMENDATIONS

In view of these facts, it is recommended:

1. That the tables of Hodges and Eyster in use since 1926, predicting the transverse cardiac diameter in the frontal plane from height, weight and age be adopted as our criteria of heart

size, with a deduction for the present of 8 mm. in the case of women.

2. That $\pm 10\%$ is a fair upper limit of the normal heart.

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DR. SCADDING—While we're all familiar with the occasional comprehensive statistical bulletins on public health questions distributed widespread over the signature of Dr. Dublin, it wasn't until last year that we had the pleasure of listening to a paper prepared by both of these essayists. However, when one does a job well, he's apt to be asked to repeat, so we'll now have the pleasure of listening to a paper by Dr. Louis I. Dublin and Mr. Herbert Marks of the Metropolitan Life.

MORTALITY OF RISKS WITH ASTHMA

By LOUIS I. DUBLIN, PH. D., THIRD VICE PRESIDENT AND
STATISTICIAN AND HERBERT H. MARKS,

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The recognition and description of asthma goes back many hundred years. Knowledge of the allergic background, however, and the principles of treatment based thereon are relatively modern developments in medicine. Allergy and its various manifestations are today being studied most intensively in many medical centers in widely scattered parts of the world and the results of these researches may eventually change the whole outlook on diseases of this kind. There are evidences of this already in the optimistic attitude of many clinicians towards the prognosis of allergic diseases.

From the point of view of insurance medicine, asthma is the most important of these diseases. There is an old saying that "asthma never kills", but in spite of that, it is a condition of which all of us have been most wary. After all the observations of even the most skilled clinicians are limited by their inability to follow up large numbers of cases and for this reason alone, we must be on our guard against their optimism. At the Metropolitan our viewpoint has changed back and forth. Formerly, we were especially afraid of young persons with the disease because of the danger of confusing it with tuberculosis. Investigation showed, however, that fear of this was largely unwarranted. The emphasis then, shifted to the danger of premature development of heart disease and, consequently, we have in recent years been more severe with the older applicants. But our caution has been such that most cases at all ages have been accepted for insurance only at substandard rates.

Our concern regarding applicants with asthma has been justified moreover by past experience. In the Specialized Mortality Investigation covering standard risks during the years 1870 to

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1900, those past age 40 were found to have a high mortality. The Medico-Actuarial Study, likewise limited to standard risks and covering the period 1885 to 1909 showed a mortality in excess of normal for asthma cases, and again it was the older risks who made the worse showing. Dr. Toulmin's investigation of the Penn Mutual's experience from 1885 to 1918 gave similar results. The experience on asthma risks was again reported on in the recent Medical Impairment Study which covered the period 1909 to 1928, and it was found that certain classes of those accepted at standard rates yielded a mortality close to that expected for a substandard group, whereas those insured on a substandard basis gave very bad results, particularly among older risks.

The seriousness of the asthmatic condition is not an easy one for the medical director to assess from the facts given by the examiner. Seldom does the latter see the applicant in the throes of an attack, but usually during a free interval. Consequently, we are forced to depend largely on the statements of the applicant who is not always frank or accurate regarding the details of his illness. We seldom can ascertain the specific offending substances which cause the attacks in each case; whether or not an attempt has been made to discover them and curative rather than merely palliative treatment given.

Hay fever, unless specified as complicating asthma, is excluded from this experience. In a sense, the two conditions should be considered together because the underlying pattern is the same. But for a variety of reasons we properly expect hay fever to have a much less effect on longevity than asthma. Hay fever is milder by and large; is usually limited, in its manifestations, to the nasal region; and is definitely seasonal in character. Asthma is caused by a much greater variety of proteins; its attacks may recur throughout the year; and its manifestations are more severe, involving characteristically the bronchial portion of the respiratory tract. This experience is limited, therefore, to cases with a history of asthma. We realize, of course, that in a number of instances, the diagnosis of asthma reported by the applicant may be incorrect.

The present paper gives the experience of the Metropolitan Life Insurance Company on 1,826 white applicants accepted for insurance during the years 1912 to 1928, inclusive, and traced to the policy anniversary in 1932. The study was made at the suggestion of the medical officers of the Company. Especially are we indebted to Dr. Jimenis who has offered us freely of his time and counsel in the various problems which came up in analyzing this experience.

The experience includes applicants showing evidence of asthma on examination and those showing no clinical signs, but reporting a history of asthma in the past. Included also is a residual group in which the medical examiner reported physical findings such as prolonged or wheezy expiration or musical rales, which in his judgment were indicative of asthma, although the applicant denied ever having it. All of these cases were free from other medical impairments or histories which would of themselves cause the applicant to be limited to substandard insurance. They all were engaged in occupations rated standard. All of them had normal hearts according to the field examiner's report.

In our analysis we have considered as far as possible the factors which might be expected to affect mortality, namely, the time elapsed since the last attack, the severity of the condition, the build of the applicant and his age. Unfortunately, the material did not lend itself to analysis according to the chief clinical types nor was it sufficiently extensive to consider the important question of occupation. We have studied the experience on males and females separately and we have combined standard and substandard cases on the assumption that they would tend to separate out when we analyzed the important factors entering into the rating. We do, however, give separately the aggregate results on the standard and substandard issues.

The aggregate results are presented in the form of ratios of actual to expected deaths by the American Men Mortality Table and the Basic Mortality Table, computed for use in the Medical Impairment Study. In the detailed groups the only standard used has been the American Men Mortality Table. On this table ratios have been computed, both on a select and ultimate basis

and on an ultimate basis, but we shall quote the ratios by the first of these. The other ratios will be found in the tables.

MALE RISKS WITH ASTHMA

The 1,503 male risks give us a total of 10,845 years of life, and there have been 99 deaths. These actual deaths were 127.4 per cent. of the expected, as measured by the American Men Table on a select and ultimate basis. The experience has been least favorable in the years immediately following issue. The ratio of actual to expected deaths has been 151.2 per cent. in the first five insurance years on the basis of select rates, but only 109.8 per cent. in the sixth and later years, using in this instance the ultimate rates.

When this experience is analyzed according to attained age, no definite tendencies are discovered. Between ages 35 and 44, the mortality was over 50 per cent. in excess of the expected by the American Men Table. The limited experience at ages 65 and over was also unfavorable, with an excess mortality of 85 per cent., but in view of the small experience at these ages, we do not place much significance upon this last figure. Strangely enough, in the preceding decennium, between ages 55 and 64, the lowest mortality ratio is found, namely, 83.3 per cent.

TABLE 1.

Mortality of Male Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. Classified by Age.

| Attained Age Groups | Years of life exposed to risk | Actual deaths | Per Cent. Actual of Expected Deaths | | | |
|------------------------|--|------------------|-------------------------------------|----------|------------------|------------------------|
| | | | Aggregate | | First 5 years | 6th and later years |
| | | | Select and Ultimate | Ultimate | Select | Ultimate |
| All Ages | 10,844.94 | 99 | 127.4 | 116.8 | 151.2 | 109.8 |
| Under 25 | 1,212.21 | 5 | 116.6 | 103.1 | * | * |
| 25-34 | 2,936.01 | 15 | 126.1 | 114.7 | 127.5 | 124.0 |
| 35-44 | 3,647.82 | 30 | 154.8 | 141.5 | 210.4 | 106.3 |
| 45-54 | 2,266.16 | 29 | 127.6 | 117.4 | 135.8 | 123.0 |
| 55-64 | 700.67 | 13 | 83.3 | 76.2 | * | 85.7 |
| 65 and over | 82.07 | 7 | 184.7 | 180.4 | * | * |

*Not significant because of small number of deaths.

By the Basic Mortality Table the mortality of these male asthma cases was 163.7 per cent. of the expected. By this standard, too, the ratios of actual to expected deaths in the early insurance years are higher than in the later years, namely, 194.0 per cent. for the select period and 141.2 per cent. for the sixth and later years, respectively. These figures stamp applicants with asthma as largely of substandard calibre.

Some irregularity is again found in the specific age mortality ratios computed on the Basic Table. Four groups of ages at issue were distinguished: 15 to 29, 30 to 39, 40 to 49 and 50 and over. The highest ratio of actual to expected mortality, 188.7 per cent., was recorded for applicants between 30 and 39 at issue. The ratios in the other three groups were approximately the same, ranging from 145.4 per cent. at ages 50 and over to 160.1 at ages 40 to 49. It should be remembered, however, that the Basic Table is severer at the younger ages, but more liberal at the older ages, as compared with earlier mortality standards.

TABLE 2.

Mortality of Male Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the Basic Mortality Table.
Classified by Insurance Years and Ages at Issue.

| Insurance Years; Age at Issue | Years of Life Exposed to Risk | Actual Deaths | Per cent. Actual of Expected Deaths |
|----------------------------------|----------------------------------|------------------|--|
| By Insurance Years | | | |
| All Years | 10,785.94* | 99 | 163.7 |
| 1-5 | 5,912.68 | 50 | 194.0 |
| 1-2 | 2,627.40 | 21 | 223.6 |
| 3-5 | 3,285.28 | 29 | 177.0 |
| 6 and over..... | 4,873.26 | 49 | 141.2 |
| 6-10 | 3,499.11 | 38 | 167.0 |
| 11-20 | 1,374.15 | 11 | 92.1 |
| By Ages at Issue | | | |
| All Ages | 10,785.94* | 99 | 163.7 |
| 15-29 | 4,128.70 | 20 | 155.8 |
| 30-39 | 3,597.67 | 30 | 188.7 |
| 40-49 | 2,351.99 | 31 | 160.1 |
| 50 and over..... | 707.58 | 18 | 145.4 |

*Excludes ages under 15.

EFFECT OF TIME INTERVENING SINCE LAST ATTACK

We have distinguished three groups according to the time elapsed at examination since the last attack of asthma, namely, less than one year, one to five years and five years or over. Those applicants with the more recent attacks, most of whom are presumably active cases, had the highest mortality—141.7 per cent. by the American Men Table. The mortality experience in this group was exceptionally heavy in the first five years during which the ratio of actual to expected deaths was 172.7 per cent., compared with 117.8 in the succeeding years. The two other groups showed much lower mortalities. The ratio of actual to expected deaths was 88.4 per cent. for those whose last attack occurred from one to five years before examination, and 91.6 per cent. for those reporting a still longer interval. In both these groups, also, the ultimate experience was better than the select, but the groups are too small to place much reliance on the actual ratios obtained.

Beside these three groups, there was the residual class, previously described, of applicants who denied any history of asthma despite chest conditions, which the Company's examiner judged to be evidence of asthma. There were only 68 such cases, but there were seven deaths in this small group, compared with only 2.81 expected by the American Men Table, or a ratio of 249.1 per cent. The result is interesting, but too much significance should not be placed upon it because there are so few cases.

Of these several groups, only the first two, viz., those with one or more attacks within one year and those within one to five years of examination were numerous enough to yield reliable data on age differentials. In the group having the last attack within a year of examination, the mortality of younger risks has been the better in the aggregate. At attained ages under 35, the ratio of actual to expected deaths by the American Men Table was 136.7 per cent. of the expected, compared with 143.0 per cent. at ages past 35. The higher rate of the latter group is due to the excessive death rates at ages 35 to 44, especially in the select period. At ages 45 and over the ratio is only 131.3 per cent., or practically the same as at attained ages under 35. In the first five years after issue, the mortality of the older risks

is high, even after 45, when it is 169.5 per cent. of the expected, compared with 134.9 per cent. at ages under 35. In the sixth and later years, the situation is reversed, the ratio on the younger risks remaining at the same level as in the earlier experience years, whereas both at ages 35 and over and 45 and over it falls to approximately 115 per cent. of the American Men Table. The group with a history of asthma going back more than a year, but less than five years, yields a much higher mortality at the younger ages than at the ages past 35, the ratios being 130.7 per cent. and 72.8 per cent. respectively. The number of deaths, however, is too small to place much reliance on the actual difference.

TABLE 3.

Mortality of Male Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. Classified by Broad Age Groups and by Time Elapsed Since Last Attack.

| Time of Last Attack; Age Groups | Years of life exposed to risk | Actual deaths | Per Cent. Actual of Expected Deaths | | | |
|--|--|------------------|-------------------------------------|----------|------------------|------------------------|
| | | | Aggregate | | First 5 years | 6th and later years |
| | | | Select and Ultimate | Ultimate | | |
| Within 1 year | | | | | | |
| All Ages | 6,888.59 | 68 | 141.7 | 129.8 | 172.7 | 117.8 |
| Under 35 | 2,634.85 | 14 | 136.7 | 122.7 | 134.9 | 140.1 |
| 35-44 | 2,370.09 | 21 | 166.4 | 152.4 | 220.0 | 119.2 |
| 35 and over | 4,253.74 | 54 | 143.0 | 131.8 | 190.5 | 114.5 |
| 45 and over | 1,883.65 | 33 | 131.3 | 121.4 | 169.5 | 112.6 |
| 1 to 5 Years Ago | | | | | | |
| All Ages | 2,500.38 | 15 | 88.4 | 81.3 | 98.2 | 81.4 |
| Under 35 | 1,193.67 | 6 | 130.7 | 116.7 | * | * |
| 35 and over | 1,306.71 | 9 | 72.8 | 67.7 | * | * |
| 5 Years Ago or More | | | | | | |
| All Ages | 1,020.66 | 9 | 91.6 | 84.7 | * | * |
| Unknown—History denied but diagnosed by examiner | | | | | | |
| All Ages | 435.33 | 7 | 249.1 | 222.9 | * | * |

*Not significant because of small number of deaths.

THE EFFECT OF SEVERITY ON MORTALITY

We have also analyzed the experience on male asthmatic risks from the point of view of the severity of the condition. Classification according to severity presents certain difficulties. Often we have simply the bald statement of the examiner that the asthma is mild, moderate or severe. This judgment is frequently a subjective reaction of the applicant. In many others, details are lacking or are too meager to furnish us a certain basis for determining the frequency or severity of the particular case. These difficulties explain some of the inconsistencies in the results we have obtained. Dr. Jimenis drew up for us the following classification, after reviewing a large number of cases.

Classification According to Severity

1. Mild—one attack a year; not more than two weeks in duration; specified as mild without further details; or specified as an accompaniment of hay fever.
2. Moderate—two or three attacks a year or duration two to four weeks; or specified as moderate without further details.
3. Severe—more than three attacks a year or duration of more than four weeks; or specified as severe without further details.
4. Cases showing evidence of emphysema or accompanied by chronic or severe bronchitis.
5. Other and unspecified.

When details of both frequency and duration were given, corresponding to different degrees of severity, the case was classified according to the more severe of the two, e. g., three attacks lasting six weeks was taken as a "severe" case.

Only cases with the last attack within a year of examination were numerous enough to warrant study of this aspect. We found that cases with emphysema or bronchitis showed by far the highest mortality. This was a small group of only 50 cases, but there were eight deaths, as against 2.19 expected by the

American Men Table, or a ratio of actual to expected, of 365.3 per cent. The next highest ratio was in the residual group on which we had either insufficient or no details. In this group of 95 cases, there were 10 deaths and the ratio of actual to expected mortality was 223.7 per cent.

Of the other three groups, the highest mortality was recorded among cases classified as of moderate severity, for which the ratio of actual to expected deaths was 144.3 per cent. by the American Men Table. The mild cases gave a slightly better experience. The ratio of actual to expected deaths in this group was 129.1 per cent. The lowest mortality of all was recorded for the group classified as severe cases, for which the ratio of actual to expected deaths was 102.5 per cent. by the American Men Table. What this means we do not know at present. It may be that our classification of these cases is at fault or that in our extreme caution in accepting such cases, we have taken only those which were far above average in other respects. Our groups, however, are not very large at best and this may explain the lack of correlation obtained.

TABLE 4.

Mortality of Male Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. Cases with Last Attack Within One Year.
Classified According to Severity of Condition.

| Severity of Condition | Years of life exposed to risk | Actual deaths | Per Cent. Actual of Expected Deaths | | | |
|--|-------------------------------|---------------|-------------------------------------|----------|---------------|---------------------|
| | | | Aggregate | | First 5 years | 6th and later years |
| | | | Select and Ultimate | Ultimate | Select | Ultimate |
| Mild | 2,598.90 | 23 | 129.1 | 118.7 | 123.6 | 132.8 |
| Moderate | 1,082.33 | 10 | 144.3 | 130.4 | * | * |
| Severe | 2,223.32 | 17 | 102.5 | 94.4 | 143.5 | 72.8 |
| With Chronic or Severe Bronchitis or Emphy- sema | 322.59 | 8 | 365.3 | 329.2 | * | * |
| Other and unspecified.... | 661.45 | 10 | 223.7 | 203.7 | * | * |

*Not significant because of small number of deaths.

THE EFFECT OF BUILD ON MORTALITY

The build of the asthmatic is an extremely important factor in the prognosis. This observation is based on an analysis of the experience of cases insured within a year of their last attack. It was discovered that underweights made a very poor showing. The ratio of actual to expected deaths by the American Men Table was 205.3 per cent., based on 27 deaths. The mortality of overweight asthmatics was also excessive, although much better than for underweights. The mortality ratio for this group, based on 24 deaths, was 144.2 per cent. In contrast with these groups is the record of those of average weight whose mortality was only 89.0 per cent. of the expected by the American Men Table. Unfortunately, the amount of material in these several groups does not warrant analysis according to age, but there is some indication of a somewhat heavier mortality among young underweights than among the older ones. At every age, however, the underweight asthmatics make a poor showing.

TABLE 5.

Mortality of Male Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. Cases with Last Attack Within One Year.
Classified by Weight.

| Build Group* | Years of life exposed to risk | Actual deaths | Per Cent. Actual of Expected Deaths | | | |
|---------------------|-------------------------------|---------------|-------------------------------------|----------|---------------|---------------------|
| | | | Aggregate | | First 5 years | 6th and later years |
| | | | Select and Ultimate | Ultimate | Select | Ultimate |
| Underweight | 2,237.40 | 27 | 205.3 | 188.9 | 263.2 | 161.1 |
| Average weight..... | 2,689.33 | 16 | 89.0 | 81.0 | 128.0 | 59.1 |
| Overweight | 1,944.10 | 24 | 144.2 | 132.1 | 140.6 | 146.9 |

*Classification based on average weights at age 27.

Underweight—10 pounds or more under average.

Average weight—9 pounds or less under or over average.

Overweight—10 pounds or more over average.

THE FACTOR OF OCCUPATION IN ASTHMA

Unfortunately we do not have enough data to study this factor from our material, but a few remarks on this will be pertinent. First of all, many of the so-called occupational asthmas of the

past have been discovered not to be asthma at all. This applies with particular force to miners' asthma and potters' asthma. By far, the greatest hazards, as we may infer from the extensive literature, are furnished by the organic dusts, particularly of animal and vegetable origin. Asthma is a real hazard to persons with allergic tendencies who deal with such materials as felt, fur, hemp, jute, flax, linen and wood. The same may be said of similar individuals who work around animals, although such cases are much less frequent since mechanical means of transportation have so largely displaced the horse. Dusts of a less definite nature, but containing some substance to which the particular individual is sensitive, are also often responsible for asthma of the so-called occupational type. It is, therefore, extremely important in judging the desirability of an asthmatic risk to consider whether the condition is due to factors of this kind and is aggravated by exposure in his occupation.

FEMALES WITH A HISTORY OF ASTHMA

Our experience on female applicants with asthma is extremely limited. We had a total of 323 cases of all types which gave us an exposed to risk of 2,194 years of life. There were 25 deaths, or 174.9 per cent. by the American Men Table and 225.2 per cent. by the Basic Mortality Table. This experience is much worse than on males for whom the actual mortality was only 127.4 per cent. by the American Men Table. We are not inclined to stress this difference, however, because the mortality ratio on females reporting the last attack within the year preceding examination was approximately the same as for men. In the small group reporting the last attack from one to five years before examination the mortality was very high—namely, 219.0 per cent. of the expected. Among females, the experience of the select period was slightly higher than in the ultimate, the ratios being 186.8 per cent. and 163.7 per cent., respectively, on the basis of the American Men Select and Ultimate rates. The experience at ages under 35 was somewhat worse than at ages past 35, but one should not place too much reliance upon this result in view of the paucity of the data.

TABLE 6.

Mortality of Female Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. Classified by Time Elapsed Since Last Attack.

| Time of Last Attack | Years of life exposed to risk | Actual deaths | Per Cent. Actual of Expected Deaths | | | |
|-----------------------|-------------------------------|---------------|-------------------------------------|----------|---------------|---------------------|
| | | | Aggregate | | First 5 years | 6th and later years |
| | | | Select and Ultimate | Ultimate | Select | Ultimate |
| Total | 2,193.58 | 25 | 174.9 | 158.9 | 186.8 | 163.7 |
| Within 1 Year..... | 1,475.57 | 14 | 148.1 | 133.7 | * | * |
| 1 to 5 Years Ago..... | 631.07 | 9 | 219.0 | 197.8 | * | * |

*Not significant because of small number of deaths.

COMPARISON ACCORDING TO STANDARD OR SUBSTANDARD RATINGS

On the whole, the rating of these cases has been quite satisfactory. About one-third of the male applicants were granted standard insurance which represents a mortality up to 125 per cent. of normal; another third approximately, Intermediate insurance, which allows for an extra mortality of 25 per cent. to 50 per cent. of normal; and slightly more than a third, Special Class, which permits from 50 per cent. to 100 per cent. excess mortality. The proportion for female applicants was roughly the same.

Male applicants to whom standard insurance was issued gave us a ratio of 87.6 per cent. actual of expected mortality by the American Men Table and 112.0 per cent. by the Basic Table used in the Medical Impairment Study. Our substandard risks, Intermediate and Special Class combined, showed a mortality of approximately 152.0 per cent. by the former table and 195.9 per cent. by the Basic Table. Compared with the American Men Table, our Intermediate risks had a mortality 110.7 per cent. of the American Men Table and our Special Class risks, 186.1 per cent.

Despite the paucity of data on females, our results were moderately in line with expectations. The mortality of standard cases,

based upon only five deaths was 101.8 per cent. by the American Men Table and 131.2 per cent. by the Basic Table. The ratios for substandard cases were 213.9 per cent. and 274.3 per cent., respectively. Females granted Intermediate had a mortality of 168.8 per cent. of the expected by the American Men Table and those limited to Special Class, 260.3 per cent.

TABLE 7.

Mortality of Risks with a History of Asthma.

Ratio of Actual to Expected Deaths by Specified Mortality Tables.
By Sex. Standard and Substandard Cases Separately.

| Sex; Department of Issue | Years of Life Exposed to Risk | Actual Deaths | Per Cent. Actual of Expected | |
|-----------------------------|-------------------------------------|------------------|------------------------------|--------------------------|
| | | | American Men Table | Basic Table 1909-1927 |
| Males | | | | |
| Standard (Ordinary).... | 4,040.60 | 26 | 87.6 | 112.0 |
| Substandard | 6,804.34 | 73 | 152.0 | 195.9 |
| Intermediate | 3,084.63 | 24 | 110.7 | |
| Special Class | 3,719.71 | 49 | 186.1 | |
| Females | | | | |
| Standard (Ordinary).... | 772.59 | 5 | 101.8 | 131.2 |
| Substandard | 1,421.00 | 20 | 213.9 | 274.3 |
| Intermediate | 771.65 | 8 | 168.8 | |
| Special Class | 649.35 | 12 | 260.3 | |

CAUSES OF DEATH

Our experience on asthmatic cases is too small to permit any detailed study of the causes of death in the various classes according to recency and severity of attack, and for that reason, only the results on the aggregate for males are presented. Of the 99 deaths among them, 25, or almost exactly one-fourth, were from heart disease, and all but three of these from organic heart disease. Influenza and pneumonia, together, accounted for 23 deaths; tuberculosis, 11; asthma, 9; and other respiratory diseases, 3. All respiratory conditions, therefore, accounted for nearly half of the deaths.

We have made a rough approximation of the relation between the actual and expected mortality from the chief causes of death of male risks. Their heart disease mortality is about two and one-third times the normal. The particular type of heart involvement most frequently certified was myocarditis. Heart strain, therefore, resulting from continued attacks of asthma is an important factor in the high mortality of these risks.

The mortality from many respiratory conditions is excessive. That from influenza and pneumonia is exceptionally high, between two and one-half and three times the normal. This excess seems to be present at practically every age. Tuberculosis mortality is higher than normal, although not in the same degree as heart disease and pneumonia. The eleven deaths from tuberculosis among these asthmatics is between one-fourth and one-third in excess of standard. The number of deaths recorded as due to asthma—one-eleventh of the total—is far greater than normal. Moreover, there were 15 in which asthma was specified as the contributory cause.

SUMMARY AND CONCLUSIONS

Let us summarize our findings and see what conclusions may be drawn from them.

1. On male risks with asthma, the mortality in the aggregate is 27.4 per cent. in excess of the expected by the American Men Table and 63.7 per cent. by the Basic Mortality Table. The results on female risks are even worse.

2. The mortality during the early years after issue is particularly high, indicating a very short select period.

3. There is an excessive mortality at practically every age, especially between ages 30 and 50.

4. Cases suffering the last attack within a year of examination have a death rate one and one-half times as high as the American Men Table; those with a more remote history, a mortality slightly better than expected by the Table.

5. Especially high is the mortality among asthmatics whose condition is complicated by emphysema or bronchitis, and among those whose condition was diagnosed by the Company examiner

despite denials of a past history of asthma by the applicant. The grading of cases according to severity yielded substantially negative results otherwise.

6. Build is an extremely vital factor. Both underweight and overweight asthmatics have excessive mortalities—twice and one and one-half times the expected by the American Men Table, respectively.

7. Despite the extra risk involved, the actual rating of these asthmatic cases has been satisfactory on the whole.

8. The excessive mortality of asthmatics is due primarily to myocardial disease and to respiratory affections, notably, pneumonia, tuberculosis and asthma.

These mortality results as well as the findings of earlier insurance studies on asthma are in sharp contrast with the observations of many clinicians with extensive experience in treating the condition that the prognosis in asthma is excellent. As Bray strikingly puts it "many asthmatics pant on to a good old age". In the opinion of clinicians the excess mortality in insurance experiences is due to the inclusion of cases of chronic bronchitis or emphysema. Such patients, they assert, regardless of whether their condition is complicated by asthma, do badly. We cannot, of course, overlook this viewpoint, especially when we have complete details on the applicant's asthmatic history. We must not forget, however, that these physicians are in a better position than the average medical examiner in the field to secure the requisite information upon which to base an accurate diagnosis. On the other hand, we should not take the optimistic attitude of clinicians at its face value. They may be right, but their observations have not been put to the test of actual mortality tables. In dealing with the cases which come up to us for rating, with histories often unreliable, we must be guided by our mortality results on similar cases.

This mortality experience shows that asthma is a serious impairment. If the history is an old one, however, without recent recurrences, applicants are usually safe for standard insurance. On the other hand, those whose last attack occurred within a year, who presumably still suffer recurrent attacks, are pretty definitely of substandard calibre. A few may be accepted for

standard insurance if they are average weight and otherwise unexceptionable. Underweight in any appreciable degree debars asthmatics from standard insurance. Indeed, the very thin asthmatic is usually not insurable on any plan. Our overweight limits on asthmatics, too, should be more rigid than on normal persons. Evidence of bronchitis or emphysema also demands a sharp rating up of the applicant.

It is, of course, impossible to secure facts, in the individual case, on the substances to which the applicant is sensitive. In certain cases if it is possible to obtain this information from the applicant's physician, it should be done because it will be of value in determining the desirability of the risk. Especially should attempt be made to secure this information on borderline cases and also where a large amount of insurance is involved. This information taken in conjunction with the medical history of the applicant and his occupation may be decisive in rating the risk. The matter of occupational exposure is important, but we have no insurance data on the extra risk of asthma involved in certain occupations. Despite this, we should take cognizance of the results of clinical studies which show the importance of certain animal and vegetable substances and certain chemicals. The occupational factor should be considered where information on the offending substance cannot be secured, but the history indicates the probability of an occupational background of the asthma.

In conclusion, may we suggest that in view of the rapid progress being made in the treatment of allergic conditions that it would be desirable for the Association to canvass the asthma situation at some future date. By securing the cooperation of some of the large clinics treating hundreds of cases, it may be possible to solve many of the vexing questions regarding prognosis and arrive at a sounder rating basis than is afforded by the study of insurance experiences only. Such an investigation would also add much to scientific knowledge of asthma.

DR. OLD—One year ago, a paper on asthma and allied conditions was read before the Association of Life Insurance Examiners of Philadelphia by one of the most prominent and competent clini-

cians in the specialized field of allergy. In his summary, he stated that the patients who frequented his clinic were full of "pep"; successful in their businesses; made the best of citizens; and there had been no deaths. Consequently, he thought they should prove to be excellent risks for insurance and even be granted full disability.

During the discussion, some insurance statistics were quoted, and as a result of this meeting, I presented a paper before the fall meeting of the Society for the Study of Asthma and Allied Conditions, in New York City, on "Asthma from the Life Insurance Standpoint".

The Medico-Actuarial mortality investigation from 1885 to 1908, inclusive, comprising 17,792 deaths, showed an average mortality of 121 ± 5 in the four groups of asthma cases. These cases were given standard insurance. The mortality was relatively higher at the older than at the younger ages at entry. The death rate from pneumonia and respiratory infections were slightly higher than normal, and practically normal from tuberculosis. There were 16 deaths out of 274 from cardiac asthma and emphysema.

The recent Medical Impairment Study from 1909 to 1927, inclusive, comprising 41,000 deaths, showed an average mortality of 112 ± 6 in the two groups of asthma cases enumerated taken standard, and an average of 233 ± 9 in those taken substandard. The death rate from organic disease of the heart was $3\frac{1}{4}$ times the normal; from pneumonia $3\frac{1}{2}$ times the normal; and from influenza $2\frac{1}{4}$ times the normal.

Out of 186 deaths in a sample of the data, $\frac{5}{6}$ were in the class, "asthma, moderate or not specified", the balance being evenly divided between severe asthma and hay fever. In a representative section of the material, there were 16 deaths from asthma (cardiac) and emphysema against less than one expected death.

Dr. Campbell and Dr. Bolt, of the New York Life Insurance Company, investigated their experience with cases of asthma in order to compare it with the recent Medical Impairment Study, and very kindly let me have the data. The group accepted as standard was too small to show the mortality trend. The group taken as substandard showed a mortality of 270%.

The two predominating causes of death were heart disease and pneumonia, and these were practically the same.

Now we have presented to us the very extensive and thorough study by Dr. Dublin and Mr. Herbert Marks of the experience of the Metropolitan Life Insurance Company with asthma cases. It comprises the issues of 1912-1928, carried to anniversary in 1932. If any of you had any doubt about the seriousness of this impairment as regards longevity, I suggest that you thoroughly digest that experience and their analysis and summary. Only congratulatory and commendatory words can be applied for this presentation, and I must confess that I was greatly pleased to see that it confirmed my opinion that asthma cases are largely and definitely substandard. Only one-third of their cases were issued standard insurance (125% of the normal) and they are to be congratulated on the favorable mortality they obtained. Standard and substandard issues combined showed for all ages a mortality of 151.2% for the first five years, and 109.8% for the sixth and later years. On the basic table of the M.I.S. these percentages would respectively be 194% and 141.2%.

The mortality from heart disease was $2\frac{1}{3}$ times the normal, and from pneumonia and influenza $2\frac{1}{2}$ to 3 times the normal. Their tuberculosis rate was higher than normal ($\frac{1}{3}$ to $\frac{1}{4}$), whereas in the other three reports, it was about normal. There were 9 deaths for which asthma was stated to be the primary cause, and there were 15 in which it was said to be contributory. I think we can classify these as being probably due to cardiac failure.

Their summary and conclusions, in my judgment, are most comprehensive and complete, and I concur in them.

Possessing a somewhat inquisitive mind, and aware of the fact that the causes of death in an impaired group may indicate, rather more than the mortality ratios, the seriousness of the impairment as regards longevity, I naturally was struck by the main causes of death in all of the four investigations being from heart disease or respiratory infections—namely—changes in the lesser or pulmonary circulation. Today, arteriosclerosis and capillary fibrosis (not the senile degenerative type, but the com-

pensatory type on the part of nature at all ages to strengthen over-active or fatigued or weakened blood vessels) is found to be the beginning etiological factor in many diseased conditions. Such changes limit the proper blood supply to the respective affected organs and tissues, and if continued over a prolonged period of years cause anoxemia and loss of function.

I also think that it is an almost generally accepted fact that hypertension is the precursor of arteriosclerotic changes and not the sequela. Again, hypertension can be caused by a low grade toxicosis acting over a prolonged period; by some mechanical obstruction; and by a sensitized individual as regards a spasmogenic aptitude or an imbalance of the cardiovascular system. We have all three of these factors acting in cases of bronchial asthma, and in addition we have the heart muscle capable of being directly though slowly infected by any foci of infection that may be present at the bases of the lungs. Hence the terminal main causes of death in these cases may be thus adequately explained.

Consequently, I would add to the requirements as enumerated by Dr. Dublin and Mr. Marks, stereoptican X-ray plates of the chest, and also eliminate any obstruction in the nares or upper respiratory tract, and any foci of infection. I believe that hay fever and bronchial asthma are each allergic reactions, and may be exchanged the one with the other in the same individual, depending largely upon the amount and constancy of dosage, and whether or not additional factors as nasal obstructions or foci of infection occur.

I think we should drop the term "cardiac asthma" as a separate and diseased condition and look upon it as representing the syndrome of cardiac failure, or nocturnal angina, caused by pathological changes having occurred or occurring in the lesser or pulmonary circulation.

DR. SCADDING—The discussion will be continued by Dr. Alfred H. W. Caulfeild, sometime Assistant to Professor Schmorl, Dresden; Professor Wasserman, Berlin; Professor Eppinger, Vienna; Sir A. E. Wright's staff, St. Mary's Hospital, London; resident pathologist, Toronto General Hospital. He has published

many papers on tuberculosis, hay fever and asthma, and is President of the Society for the Study of Asthma, as well as Editor of the Journal of Allergy.

DR. CAULFEILD—Dr. Dublin states in his paper that "Asthma has long been recognized as a separate disease entity". I doubt that that is our conception today. Certainly it is not a separate disease as we understand it, for example, nephritis or pulmonary tuberculosis. Both of these have a definite pathology; asthma has not. Asthma comprises a group of symptoms and varied signs depending upon severity and type. When it is present in its periodical and characteristic fashion it can be regarded as a temporary physiological disarrangement rather than a definite pathological entity.

Definite pathological changes come after continuation of the asthmatic attacks and the most marked and consistently found is emphysema. This leads one inevitably to a discussion of differential diagnosis.

The correctness of the initial or field examiner's diagnosis must I feel be dealt with. Upon this depends the validity of the conclusions drawn by statistical studies given us by the author, and concurred in by Dr. Olds, who has preceded me.

If I might present the following conditions which may easily be mistaken for asthma it will, I feel, let me present to you some of the ideas I have as regards the correctness of the initial or field examination. I have divided them into 4 groups as follows:

(1) True periodical asthma at the extreme right end of the picture merges indefinitely into bronchitis with marked asthma or respiratory distress at the extreme left. For the former the diagnosis of asthma is undoubted; for the latter it is frequently difficult to decide whether one should choose the name of bronchitic asthma or just bronchitis.

(2) Bronchitis is the pulmonary diagnosis that is really the waste basket diagnosis for all the other uneliminated chronic pulmonary diseases. Particularly if considerable emphysema has developed, respiratory attacks will occur suggesting the diagnosis of asthma. Many of such cases are in reality instances of bronchiectasis or chronic pulmonary abscesses. Instillation of Lipiodol

(or other radio opaque substances) has been the means of demonstrating the unexpected frequency of bronchiectasis and chronic pulmonary abscess.

(3) True essential emphysema remains as another relatively (?) rare condition which may only with difficulty be separated from asthma. In making my notes for this paper I used the word 'rare' as regards emphysema. Owing to a recent post mortem I wonder whether it should be called 'rare'. In this particular instance I had been asked to give an opinion in 1929 on a case which was diagnosed as bronchial asthma. To summarize a long story I reported that to me it seemed a rather rare instance of extremely acute emphysema and that he was not a case of bronchial asthma in any sense of the word. X-ray showed opacities which I was inclined to regard as evidence of tuberculo-fibrosis. The man died suddenly and a post mortem was held by a pathologist who, fortunately, had learnt that I was interested in the case. Post mortem showed extreme and acute emphysema on one side and on the other side atelectasis. This was recent and was the immediate cause of death. The opacities as exhibited in the X-ray films failed to show, on microscopic examination, proof of a tuberculous etiology.

(4) Tuberculosis of a disseminated type with marked emphysema will often give rise to periodic respiratory distress in such fashion as to facilitate the diagnosis of asthma-bronchitic asthma more probably.

I have taken considerable time to very briefly outline some of the differential diagnoses of individuals who may easily be wrongly regarded as instances of asthma. To this group I am inclined to add chronic bronchial wall cancer. In other words, I am convinced that in the 1,503 male and 323 female cases of asthma analyzed by Dr. Dublin there were instances of all the conditions I have enumerated. In private practice I have had illustrations of all that I have mentioned. For years I have recorded the self made or previously made diagnosis of cases referred to me. I had only to glance through the cross index cards to realize how frequently this took place. Unfortunately I did not have time to attempt a statistical analysis.

In a rather hurried survey of my own cases I found there were 6 known deaths, i. e., those which took place while under observation or shortly afterwards. They might be summarized as follows:

Two cases were essentially cardiac. In one, respiratory distress appeared and it was very suggestive of an allergic sensitization. Skin tests proved this and the clinical picture was considerably modified by deletion of the factors to which the case was sensitive. In due course of time death took place on account of the cardiac condition. The second case had exhibited essentially high blood pressure for years with the development of hypertrophy of the heart. The first "attack" was regarded as cardiac though the suspicion of allergic factors must have come in as a few skin tests were carried out. By what might be regarded as almost an accident adrenalin was administered with the marked benefit one associates with an allergic asthma rather than a cardiac condition. Within two years the case died and the clinical picture was essentially that of myocarditis with a terminal exacerbation of a co-existent nephritis. Except for some of the attacks which could only be relieved by adrenalin there was no suggestion of any true allergy. The third case was definitely that of asthmatic bronchitis. The respiratory distress was in my opinion entirely due to the amount of emphysema. There were no personal or hereditary factors suggesting allergy. The case died but no post mortem was held. The fourth case came with a definite diagnosis of bronchial asthma both by the patient and his doctor. There had been four years of this type of respiratory distress suggesting asthma. The case in my opinion did not suggest asthma and the few skin tests done were only a preliminary and because they were almost demanded. Fuller investigation showed bronchiectasis which later was shown to be associated with bronchial wall cancer. The fifth case had a positive allergic history, both personal and hereditary. Appropriate skin tests showed sensitization to pollens and other factors. There was also present a bronchiectasis which latter was probably the indirect cause of death. The sixth case was the only one which was, in my opinion, a true allergic asthma and this individual died in an attack.

Of this group one certainly, and possibly two, justified the diagnosis of bronchial asthma. Five of them certainly were bad risks. All of them were over or near 50 years of age.

Of really characteristic asthma there can be no doubt. Outside of this group one is frequently in doubt as to whether the word asthma should be used, especially when we actually don't know what asthma really is. Just to say it is a result of spastic contraction of the bronchial wall musculature or, if you prefer, engorgement of the bronchial vessels and oedema or both doesn't get us very far. Not even in the characteristic case, for which we can demonstrate the cause, and relieve or induce an attack by the exhibition or withdrawal of the cause, are we advanced very far in a fundamental understanding of the condition (I prefer that word to disease).

It is perhaps not going too far to state there is no definite pathology known which can consistently be demonstrated at post mortem—except the varied type of the induced emphysema. I have seen several post mortems on cases dying in a true attack. The type of emphysema has varied in all. There is no consistency in the post mortems reported in the literature.

(3) I feel certain that this is the explanation of the frequent findings of tuberculosis as the cause of death. Time prevented me giving actual figures on cases referred to me as asthmatics who really had tuberculosis.

(4) On general clinical experience I would have ventured to put a high mortality between 40 and 50 whereas the statistical findings put it at between 30 and 50.

(5) Unquestionably on clinical experience one would expect a high mortality with cases complicated by marked emphysema or bronchitis.

I congratulate Dr. Dublin on his statistical tables and his conclusions based upon those tables. On his material I am inclined to concur but I feel certain his material included more individuals suffering from other diseases than it did those who should actually be diagnosed as asthma. With Dr. Olds I agree that the diagnosis of cardiac asthma has to be proved before its relatively rare acceptance is justified.

The varied means required for the differential diagnoses of cases of asthma or suspected as being instances of asthma extend of course beyond the means of field or routine life insurance examinations. I do feel, however, that a logical suspicion of this could be obtained by a carefully worded interrogation. I have looked through two or three of the forms that are filled out by the applicant and field examiner for insurance and, in my opinion, they all fail to put the questions which, if answered to the best of the applicant's knowledge, would lead one to suspect this condition and to institute subsequent and fuller interrogation with possibly, in a selected number, the employment of the appropriate methods of differential diagnosis.

The point I wish to make from this is that the cases analyzed by Dr. Dublin must have included false diagnoses. This it would be, my impression is, the explanation of (1) The particularly high mortality experienced during the early years after issue.

(2) I would be inclined to attribute the high mortality found to be due to myocardial disease on the grounds that in the beginning the individual had cardiac disease and not that his asthma induced a subsequent myocarditis.

DR. SCADDING—Dr. Dublin, have you anything to say in closing?

DR. DUBLIN—Only this, Dr. Scadding: It is perfectly obvious that no one better than Dr. Caulfeild is able to indicate the difficulties involved in this term. We are quite aware of all the troubles. It is true that under present conditions with the limitations that prevail in the field in regard to the making of accurate diagnosis, we in the Home Offices must be guided by these very broad and general results. Otherwise, we would get in all kinds of trouble. It is almost impossible to make the type of differential diagnosis you have in mind.

DR. SCADDING—At many of our meetings in the past we have been both mystified and intrigued by our next essayist's ingenious little contrivances to anticipate the machine age in urinalysis. He and his able assistant, Dr. Anton R. Rose, have collaborated on a subject rather new to these meetings.

THE DETERMINATION OF HEMOGLOBIN
IN LIFE INSURANCE SELECTION

By WILLIAM G. EXTON, M. D. and ANTON R. ROSE, PH. D.
The Prudential Insurance Company of America

Proposers for life insurance often present some doubtful or obscure feature of examination or history of previous operation or illness which the medical director is at a loss to evaluate correctly because the available information is too indefinite or insufficient. It also happens that the personal appearance and physical examination of many of these individuals, who suffer from diseases that inevitably abbreviate life, appear satisfactory to examiners.

Typical of such cases as they come for consideration are underweights with or without tubercular family history, florid overweights, unhealthy looking people with negative physical examination and history, cases in which there may be reason to suspect tuberculosis, syphilis, malignancy, polycythemia or anemia, cases with a history or finding of albumin, casts, abnormal blood pressure or heart murmurs, cases with a history of rheumatism, arthritis or digestive troubles and people who have undergone some abdominal operation other than hernia or appendicitis. Undoubtedly, any guide to improved selection among these and other questionable cases will prove welcome and valuable.

During the past few years an increasing demand on the Prudential Longevity Service for hematological examinations has focused our attention on a difference between clinical and insurance practises which is definite enough to cause us to wonder why medical directors make so little, if indeed any use at all of diagnostic information which clinicians consider fundamental.

In the clinic, the hemoglobin content of blood is determined oftener than any other constituent of the body because clinicians regard it in the nature of a test of the whole organism. All of the chronic diseases sooner or later cause secondary changes in

blood which alter its hemoglobin content, as do, of course, the diseases which primarily affect blood. As is also well known, the hemoglobin content of a blood measures its oxygen transport, which, in turn, influences every phase of an individual's metabolism.

In the course of efforts to improve our hematological technics we have, from time to time, determined the hemoglobin contents of blood from applicants for life insurance which came to the laboratory for sugar tolerance, Wassermann and other tests. While this material is not yet ample or old enough, and too little is known about the symptomatology to justify treating it statistically or clinically, the number of deviations from the normal range which were found proved to be more than sufficient to indicate clearly that hemoglobin determinations should enable a better judgment of the quality of many proposed risks presenting doubtful or obscure features of examination or history.

Recent advances in specific therapy have greatly stimulated clinical interest in the differential diagnosis of the anemias. This, of course, depends upon hematological technics for obtaining exact information concerning the number and size of the red blood cells and their saturation with hemoglobin. Without such information it is impossible to derive ratios or indices such as color, volume, saturation, etc. which are necessary for correctly distinguishing between the different kinds of anemia.

In life insurance work it is impracticable to derive such information because there is as yet no way known to preserve blood longer than four days under mailing conditions so as to secure reliable counts and volume determinations. These differentiations are, however, unnecessary in life insurance work because its purposes will be well enough served if we go along with the clinician only, so far as the point where he stops to generalize by calling all excesses of hemoglobin polycythemia and all deficiencies, anemia. That far the medical director can easily go.

HEMOGLOBIN

Although it does not lie within the scope of our present plan to go into the details, it must be confessed that it is actually

impossible to touch on the subject of hemoglobin without colliding with the differences in methods, conflicting data and varied standards that plague American hematology by causing unsettlement and confusion in all of its branches,—physiological, pathological, and technical.

Thus, in addition to the absolute values for hemoglobin, clinicians also need to know the hemoglobin content relative to normal, the hemoglobin content relative to the number of red blood cells and the hemoglobin content relative to the size or volume of the red blood cells. At the present time the clinician must get along as best he can with little or no uniformity about such information, because the authorities disagree on methods, on the normal standards, and also on the variations that may result from such collateral influences as climate, altitude, exercise, diet, time of day, etc.

The position of the medical director is fortunately otherwise, however, and it enables him to avoid many of the standardizing difficulties which clinicians have to contend with if he stops at the point where it is easy for him to confine his attention to the absolute terms of grams of hemoglobin per 100 cc. of blood. Then, all bloods having a hemoglobin content below the minimum of the normal range may be taken to definitely indicate anemia, and bloods having a hemoglobin content above the maximum of the normal range, polycythemia.

Normal standards are, of course, established by correlating the results obtained by examining many healthy people by some predetermined satisfactory method. Owing to its fugitive nature, there is no direct method for measuring hemoglobin available which is sufficiently accurate and reliable to be rated satisfactory. It is, therefore, necessary to depend upon indirect methods. Of these, the measurements of oxygen capacity or of iron serve well enough for standardizing purposes, but are too laborious and exacting for practical work.

To measure the amount of oxygen set free from its unstable combination with hemoglobin, the apparatus and method of Van Slyke is capable of a satisfactory degree of accuracy in the hands

of a well-trained chemist who is familiar with the quirks and pitfalls of gasometric technics.

To measure the iron in the chromogen part of the hemoglobin molecule, with satisfactory accuracy, there are several methods at our disposal which give results that should correspond with accurate oxygen capacity measurements, and which are, for very good technical reasons, more suitable for clinical laboratories than oxygen capacity measurements. For hemoglobin standardization, the Prudential Laboratory employs a simple and reliable iron method which it has developed for the purpose, and, accordingly, regards 14 to 17 grams of hemoglobin per 100 cc. of blood as the limits of the normal range of hemoglobin for adults. In some, particularly southern sections of the country, where it is suspected that red cell counts run lower than in other localities, it may not be amiss to shade the lower limit in the case of women, but no matter where situated, hemoglobin contents above 17 grams suggest polycythemia.

A number of authors have published data giving the normal values of hemoglobin at the different ages, but examination of the data shows that between the ages of 17 and 70 the differences are within the experimental error of all of the hemoglobin methods. The 14 to 17 gram normal range, therefore, applies to all adults likely to come within the scope of insurance selection.

The published data concerning the diurnal variations of hemoglobin are very conflicting. Thus Rabinowitch's data indicate very considerable variations, while the data of Smith and others indicate that the diurnal variations in normals are negligible. It is possible that differences in the choice of methods and human material account for the disagreement and also for the conflicting results which are similarly evident among the data available on other variations such as climate, exercise, etc.

Clinicians speak of any reduction of hemoglobin content below the normal as anemia, and in a loose practical way classify the anemias as primary, secondary, or hemolytic. In the latest text on hematology, Pepper and Farley say that the primary anemias are of "mysterious origin" and they call attention to the fact

that the outstanding clinical type of primary anemia, usually known as pernicious anemia, can no longer properly be regarded as being either primary or pernicious. They therefore call the disease Addisonian anemia and treat as individual types all of the other varieties of anemia which are neither Addisonian nor secondary. For life insurance purposes it makes little difference whether an anemia be primary or secondary because the mere finding of anemia in an otherwise doubtful or obscure case suffices to stamp it unfavorably.

In passing, it is, nevertheless, worth noting that while pernicious or Addisonian anemia has not hitherto come within the purview of life insurance selection, it will eventually have to be reckoned with because of the remarkable change in the course of the disease that has come about through advances in therapy. Instead of running its former more or less rapidly fatal course, Addisonian anemia generally responds so promptly to specific treatment that cases may now appear before examiners without showing any evidence of the disease that is discoverable by the usual insurance examination. It is also interesting to note that although they may not show sugar in the urine on examination, cases of pernicious anemia are apt to give about the same kind of responses to sugar tolerance tests as diabetics do. In fact, it has been reported that cases of pernicious anemia which have responded favorably to liver treatment show a distinct tendency to develop diabetes later.

In contrast with the so-called primary anemias, some of the secondary anemias exhibit a decidedly increased tolerance for sugar. According to the general understanding, the secondary anemias result from either the loss of blood or from a depressed or defective blood formation. Thus ulcers and other intestinal troubles, as well as many other types of disease, are often regularly accompanied by repeated small, perhaps occult, hemorrhages which depress the hemoglobin content below normal. It is also true that all but the most trifling infections tend to lower the hemoglobin content, and that anemia occurs regularly and often persists long after convalescence from many prolonged but not necessarily severe infections. Anemia also

occurs very early in some forms of nephritis, and later in others. Its presence or absence should, therefore, be helpful in appraising the significance of many albuminurias.

The tendency to anemia is always constant in focal infections. In fact, anemia is a regular consequence of toxic absorptions, whatever their source, and may even be severe in low grade long protracted intoxications exhibiting little or no symptomatology. The secondary anemias resulting from prolonged living on unbalanced diets, or for shorter times on diets deficient in iron, must also often be reckoned with as well as those produced by intestinal and other parasites. In fact, there seem to be few disease processes which do not affect the hemoglobin content of blood.

Besides the so-called primary and secondary anemias, there is another indefinite group of anemias which are conveniently called hemolytic because the term characterizes the view held by clinicians that the predominating feature of this group is neither a specific digestive cause nor a failure of the blood forming functions, but an increase in the rate of destruction of the red blood cells within the circulation.

Under physiological conditions it is reckoned that the red blood cells function from 30 to 100 days before breaking down. In this breaking down process the iron remains for re-synthesis while the rest of the hemoglobin enters into the formation of bile pigments. In hemolytic anemia this breaking down process goes on faster than in health. If the destructive process be severe enough, bile pigments will be formed in sufficient amounts to cause the jaundice which is so characteristic of some types of hemolytic anemia. While the jaundiced cases are not likely to be of practical interest in insurance selection, it is well to remember that a considerable degree of jaundice often exists without coloring the skin or conjunctiva enough to be apparent. There are also other varieties of hemolytic anemia which never have jaundice, but all of the hemolytic cases are apt to excrete urobilin or urobilinogen in the urine. Both of these pigments are derived from the bilirubin which is formed from the destroyed blood cells and both pigments can be detected and

measured by the same simple test which it is, therefore, well to apply whenever the possibility of anemia is suspected.

The hereditary and congenital anemias belong in the hemolytic group, as do also the anemia of pregnancy, the anemias caused by poisonings from certain chemicals like lead, and the anemias that result from certain infections such as malaria and syphilis, the Welch Bacillus, etc. While the above crude classification of the anemias roughly represents the usual clinical perspective, it is, of course, understood that the majority of cases are probably mixed types.

Whenever the number of red cells in the blood count is higher than normal, the hemoglobin content tends to exceed the maximum of the normal range instead of being subnormal as in the anemias. It is, therefore, usual for clinicians to call such conditions polycythemia when the red blood cells count 6,000,000 or more. Primary polycythemia or erythremia has no practical significance in life insurance selection, although I have seen two claims of cases that certainly had the disease when they were examined for life insurance.

On the other hand, the relative and secondary polycythemias are encountered in life insurance work much oftener than suspected. In fact, I have, within the last month, seen bloods that indicated polycythemia from two out of nineteen applicants for insurance whose hemoglobin contents were determined. One of them showed 18.5 and the other over 20 grams of hemoglobin per 100 cc. The secondary polycythemias result from the effects of certain chemicals, from all of the conditions associated with decreases in plasma volume, and increases in blood concentration, such as sweating, diarrhea, polyuria, water starvation, etc. They also accompany the dehydrations resulting from acidosis and other causes. Any of the many conditions which interfere with the supply or efficient utilization of oxygen, such as altitude and heart and lung troubles, also cause polycythemia, and it is interesting to note that the blood pressure often rises as a result of increases in the viscosity of the blood which so often characterize the polycythemias.

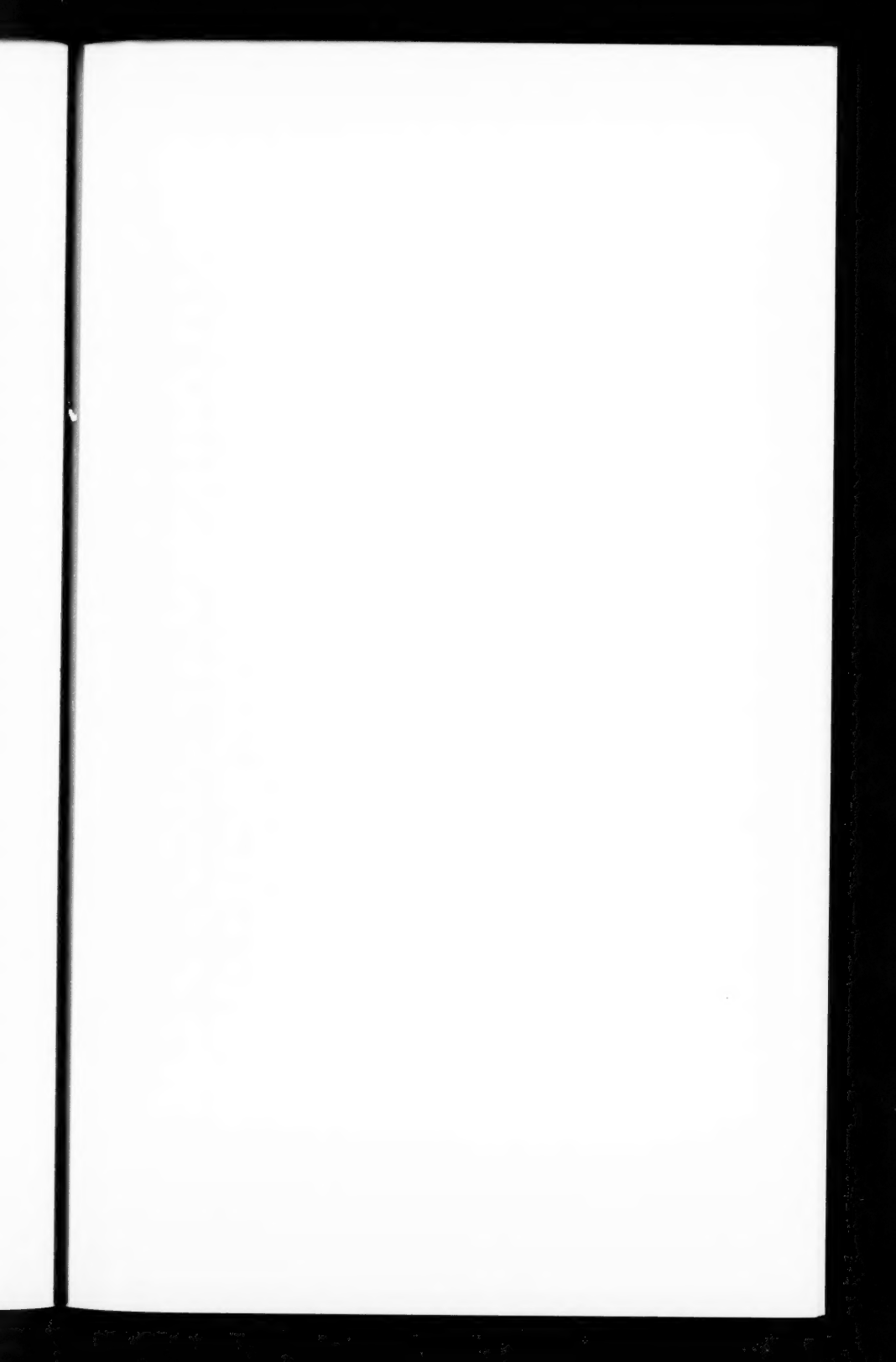
Although the foregoing summary of conditions which influence the hemoglobin content of the blood is necessarily sketchy and incomplete, it will serve to make it plain that changes from the normal hemoglobin are to be expected in a large number and variety of health conditions which may affect applicants for life insurance who present doubtful or obscure features of history or examination. Finally, it may not be amiss to point out that in judging these cases no importance or significance should be attached to a normal hemoglobin content. On the other hand, an abnormal hemoglobin content in an applicant who presents some otherwise doubtful or obscure feature marks the case a definitely undesirable life insurance risk.

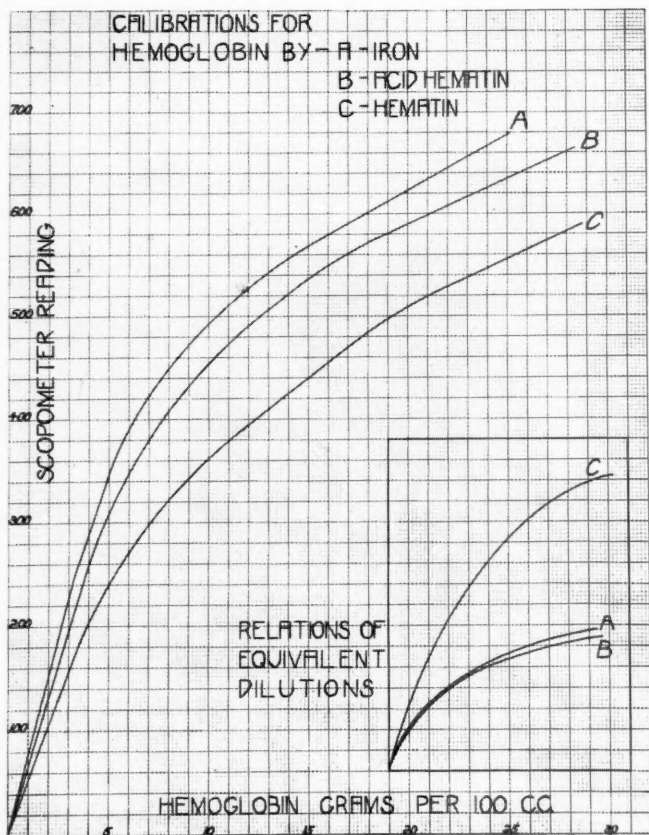
TECHNICS

Two to four drops of blood from a punctured finger tip or ear lobe suffice for the determination of hemoglobin. It is best to collect the blood in a small glass or hycoloid vial containing some dry anticoagulant like sodium fluoride and shake the vial containing the fresh blood vigorously from side to side for a moment in order to prevent clotting. Blood collected in this way will give satisfactory hemoglobin determinations after months if the acid hematin method in common use or the newer hematin method developed in the Prudential Laboratory be employed for estimating the hemoglobin.

To carry out the acid hematin method, dilute a 0.1 cc. sample of blood with 1/10 N hydrochloric acid. The amount of acid used for diluting the blood will differ with the particular instrument employed to measure the color of the resulting brownish colloidal solution. Whatever the instrument, it is essential to calibrate it to give results that correspond with either the iron or the oxygen capacity method, and whatever the dilution, the test must be allowed to stand at least half an hour at room temperature so that the color to be measured can completely develop.

To carry out the hematin method: a 0.1 cc. sample of blood is mixed with 10 cc. of 1/10 N sodium hydroxide and allowed to stand 20 minutes or longer. 2 cc. of this material is then mixed with 18 cc. of the hematin reagent. This is made up of





28 cc. of 25% sodium acetate and 56 cc. of 1/10 N acetic acid diluted to 500 cc. with distilled water. The resulting reddish precipitate is ready for measurement immediately after the test tube has been inverted gently once or twice. The accompanying chart shows the calibrations for the iron, acid hematin, and hematin methods obtained with the Photo-Electric Scopometers employed in the Prudential Laboratory for all colorimetric and turbidimetric measurements. The insert on the chart shows the threefold increased sensitivity of the hematin over the other methods.

DR. CLARK—I would like to ask Dr. Exton whether he has made any attempt to have slides made in connection with hemoglobin tests. If we were able to obtain these they might be very enlightening. Has that been taken into consideration?

DR. EXTON—We have never used blood slides in connection with insurance work.

DR. SCADDING—The next paper on our program will be presented by Dr. Charles P. Clark, a Medical Director of the Mutual Benefit Life Insurance Company.

A THEORETICAL STUDY OF BLOOD
PRESSURE AND ITS RELATION
TO HEART SIZE, BODY
SURFACE AREA AND
METABOLIC RATE

By CHARLES P. CLARK, M. D.

Mutual Benefit Life Insurance Co., Newark, N. J.

That there is a definite increase in the height of the average blood pressure with each successive decade of age has been definitely proved by numerous investigators. The relative accuracy of the tables of average pressures for age, developed from the study of insured risks presented by Fisher, Hunter, Rogers, Symonds and others, has been acknowledged both by the insurance fraternity and clinicians, and these tables have served as useful guides in the differentiation between the normal and pathological. The diagnosis of hypertension and hypotension requires a definite statement of the average, and the blood pressure of the patient or insurance applicant is usually compared to the average for the attained age as noted in an accepted table.

Why should there be this increase in the average blood pressure with advancing years? Mosenthal, in discussing the increase of pressure after age 20, states, "It may be considered that the blood pressure of 120/80 for age 20 is the 'normal pressure' for adults. The rise in subsequent years may be regarded in two ways, either that it is the physiological expression of advancing years, or that it is a sign of physical deterioration. My own conception of the situation," states Mosenthal, "is that the normal adult pressure is about 120/80 and that any rise above this signifies some change in the body, though it may be nothing more than the adjustment that comes with advancing years, yet it is to be regarded as the expression of pathological changes either functional, or anatomical, or both, that

react to the disadvantage of the individual." This explanation for increasing blood pressure after age 20 is justified to some extent but it fails to explain the marked increase which certainly occurs from birth to age 20. That the blood pressure is to be regarded as an index of vascular elasticity has been stressed by others, while the changes in peripheral resistance secondary to alteration in the arterioles has been cited as a probable factor.

If we are dealing with the subject of blood pressure deviations in disease, we must consider the influences of many pathological changes; for example, those found in arteriosclerosis, chronic interstitial nephritis, acute or chronic intoxications as illustrated by the toxemia of pregnancy or toxic goitre. In the study of blood pressure of normal subjects, however, the writer believes that we can find an explanation for the increase with age by referring to a fundamental definition of physics. From the chapter on the Mechanics of Liquids in "Practical Physics" by Black and Davis, I quote:

"Force and pressure.

In studying liquids we must distinguish carefully between force and pressure. FORCE means a push or pull. Forces are usually expressed in pounds or grams, or kilograms. PRESSURE means the push or pull per unit area of the surface acted upon. That is,

$$\text{Pressure} = \frac{\text{force}}{\text{area}}$$

This formula may be adapted to our present study by the expression:

$$\text{BLOOD PRESSURE varies with } \frac{\text{Heart Force}}{\text{Body Surface Area}}$$

"Heart force", for the study of normal individuals of various ages, we find, may be expressed in grams of heart weight. "Blood pressure", may be expressed in terms of millimeters of mercury as customarily recorded and referring to the pressure over the brachial artery.

The writer, in a separate study, devised the following index of surface area which has proved to be satisfactory for the purpose of several investigations:

"Body surface area varies with the square root of the product of height and weight"; i. e.,

$$S. A. \propto \sqrt{W \times H} \quad (\text{Note 1})$$

We may now state that:

$$\text{Blood pressure varies with } \frac{\text{Heart Weight}}{\sqrt{\text{Weight} \times \text{Height}}}$$

We may, therefore, develop an index which, from our study, we find may be considered a constant which we will call $K_{(1)}$.

$$K_{(1)} = \frac{\text{Heart Weight}}{\sqrt{\text{Weight} \times \text{Height}} \times \text{Blood pressure}}$$

With this constant we will later make a study of heart weight, but we will make a preliminary study of blood pressure by substituting Body Weight for Heart Weight in the above formula, and thus we develop a second constant, $K_{(2)}$.

$$K_{(2)} = \frac{\text{Body Weight}}{\sqrt{\text{Weight} \times \text{Height}} \times \text{Blood pressure}}$$

For the determination of theoretical blood pressure values our problem is much simplified by the substitution of body weight for heart weight, and a table thus prepared is of practical interest. From two sources we have secured sufficiently representative data to derive the numerical value of " $K_{(2)}$ " in the above formula. We have taken the average systolic pressures for the quinquennial ages 15 to 55 years proposed by Mr. Arthur Hunter, of the New York Life Insurance Company. The average height we have assumed to be 68 inches; the weight for each age at 68 inches we have assumed to be identical with that given in the weight table developed by the Medico-Actuarial Association. With this data we have drawn up the following table and have determined that $K_{(2)} = 0.01208$.

TABLE I

| Age | Height | Weight *1 | Blood pressure *2 | $K_{(2)} = \frac{\text{Weight}}{\sqrt{W \times H} \times (\text{B.P.})}$ |
|----------|--------|-----------|-------------------|--|
| 15 years | 68" | 134 lbs. | 113 m.m. | 0.01242 |
| 20 " | 68" | 144 " | 120 " | 0.01212 |
| 25 " | 68" | 149 " | 122 " | 0.01214 |
| 30 " | 68" | 152 " | 123 " | 0.01216 |
| 35 " | 68" | 155 " | 124 " | 0.01217 |
| 40 " | 68" | 158 " | 126 " | 0.01212 |
| 45 " | 68" | 160 " | 128 " | 0.01200 |
| 50 " | 68" | 161 " | 130 " | 0.01183 |
| 55 " | 68" | 163 " | 132 " | 0.01177 |

Average 0.01208

(*1—From table of average weights by Medico-Actuarial Investigation.

*2—From table of average blood pressures by Mr. Arthur Hunter.)

By the use of this numerical value for $K_{(2)}$ we may now estimate the theoretical blood pressure for any height-weight combination desired by means of the formula:

$$\begin{aligned} \text{Blood pressure} &= \frac{\text{Weight}}{\sqrt{H \times W} \times K_{(2)}} \\ &= \frac{\text{Weight}}{\sqrt{H \times W} \times 0.01208} \end{aligned}$$

In the next table we will test the accuracy of the formula by developing the theoretical blood pressure values for infancy, childhood and quinquennial ages 15 to 55. We will also compare these results with the data published by MacLaren in his volume entitled "Medical Insurance Examinations" and concerning which data that author states:

"We have summed up the 'readings' of the best observers in England and America, and the resulting figures came so near to those similarly collated by Halls-Dally, being perhaps a millimetre or so lower, that we give his table, which has the advantage of omitting decimals, and probably is compiled from a more exhaustive collation than ours. To it we have

added infantile pressures taken from Friedlander and our own experience."

The values of height and weights for childhood are taken from Holt's "Diseases of Childhood". For ages 20 to 55 the weights are taken from the Medico-Actuarial Table. In column IV are given the calculated blood pressure values, and in column V the systolic averages published by MacLaren.

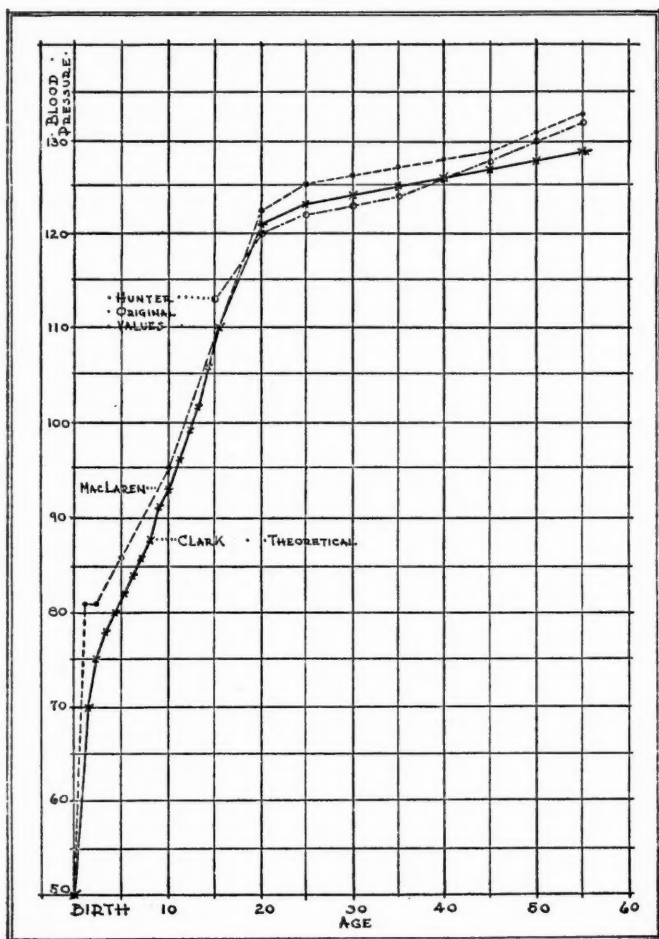
TABLE II

| Age | Average Height | Average Weight | B.P.= | Weight | MacLaren Averages Systolic |
|---------|----------------|----------------|-------|------------------------------------|-------------------------------|
| | | | | $\sqrt{W \times H \times K_{(s)}}$ | |
| Birth | 20.6" | 7.55 lbs. | | 50 m.m. | 50 m.m. |
| 1 year | 29.0" | 20.5 " | | 70 " | 81 " |
| 2 years | 32.5" | 26.5 " | | 75 " | 81 " |
| 3 " | 35.0" | 31.2 " | | 78 " | |
| 4 " | 38.0" | 35.0 " | | 80 " | |
| 5 " | 41.7" | 41.2 " | | 82 " | 86 " |
| 6 " | 44.1" | 45.1 " | | 84 " | |
| 7 " | 46.2" | 49.5 " | | 86 " | |
| 8 " | 48.2" | 54.5 " | | 88 " | |
| 9 " | 50.1" | 60.0 " | | 91 " | |
| 10 " | 52.2" | 66.6 " | | 93 " | 95 " |
| 11 " | 54.0" | 72.4 " | | 96 " | |
| 12 " | 55.8" | 79.8 " | | 99 " | |
| 13 " | 58.2" | 88.3 " | | 102 " | |
| 14 " | 61.0" | 99.3 " | | 106 " | |
| 15 " | 63.0" | 110.8 " | | 110 " | 110 " |
| 20 " | 68.0" | 144.0 " | | 121 " | 123 " |
| 25 " | 68.0" | 149.0 " | | 123 " | 125 " |
| 30 " | 68.0" | 152.0 " | | 124 " | 126 " |
| 35 " | 68.0" | 155.0 " | | 125 " | 127 " |
| 40 " | 68.0" | 158.0 " | | 126 " | 128 " |
| 45 " | 68.0" | 160.0 " | | 127 " | 129 " |
| 50 " | 68.0" | 161.0 " | | 128 " | 131 " |
| 55 " | 68.0" | 163.0 " | | 129 " | 133 " |

In GRAPH I is beautifully displayed the close similarity of the theoretical values when compared to the actual averages

GRAPH I

Calculated blood pressure values compared with Hunter's original and also MacLaren's compiled averages. (See Text and Table II.)



compiled by MacLaren. This is the more remarkable when we realize that our constant was developed with average values for adults presented by Hunter; that the average heights and weights for children were compiled by Bowditch and others over 40 years ago; that the heights and weights of adults were developed by the Medico-Actuarial Investigation and that no correction was made for weight of clothing. The data was therefore not homogeneous and the table is truly synthetic in its development.

Interesting though this formula be, it is probably of less importance than Formula I, by means of which we may study heart weight. We must first note that according to Holt—except at age 7—the ratio of heart-weight to body-weight remains practically constant during growth. However, according to Mueller, the heart-weight body-weight ratio in adults diminishes as we pass from underweight to overweight. This is well demonstrated in the following table taken from Mueller's averages:

TABLE III

| Body Weight Kms. | Mean Age | Absolute Heart Weight Grams | Ratio of Heart-weight to Body-weight |
|---------------------|----------|--------------------------------|---|
| 30-40 | 51 | 193.3 | 0.00547 |
| 40-50 | 51 | 230.2 | 0.00510 |
| 50-60 | 49 | 264.3 | 0.00481 |
| 60-70 | 51 | 297.2 | 0.00445 |
| 70-80 | 59 | 322.3 | 0.00437 |
| 80-90 | 44 | 359.0 | 0.00428 |
| 90-100 | 56 | 376.3 | 0.00401 |
| 100-110 | 54 | 358.5 | 0.00346 |

It appears probable from the study that follows that heart weight varies with the product of surface area and blood pressure.

If we will return to our formula

$$\text{Blood Pressure} = \frac{\text{Heart weight}}{\text{Body surface area}}$$

$$\text{and } K_{(1)} = \frac{\text{Heart Weight}}{\sqrt{W \times H \times \text{B.P.}}}$$

we can then develop the value of $K_{(1)}$ for ages 50 to 55 by means of Mueller's normal heart values, given above, with body weight translated into pounds:

TABLE IV
Mueller's Normal Heart Weights
(Ages 44-56)

| Body weight in pounds | Heart weight in grams |
|-----------------------|-----------------------|
| 80 lbs. | 193.3 |
| 100 " | 230.2 |
| 121 " | 264.3 |
| 148 " | 297.2 |
| 163 " | 322.3 |
| 194 " | 359.0 |
| 207 " | 376.3 |
| 229 " | 358.5 |

Average 155 lbs.

300.0 grams

We may assume that the above weights were taken without clothing and that the average height, without shoes, was approximately 67 inches. We shall also assume that the average blood pressure was approximately that given by Hunter and Symonds for ages 50 to 54 = 130 m.m.

$$\text{Then } K_{(1)} = \frac{300}{\sqrt{155 \times 67 \times 130}} = 0.02265 \quad (\text{Note 2})$$

With this value of $K_{(1)}$ we can now estimate the heart weight for any height-weight combination, providing we have the corresponding blood pressure value. For example, at birth:

Average height = 20.6" (Holt)

Average weight = 7.55 lbs. (Holt)

Average B. P. = 50 m.m. (MacLaren)

$$\begin{aligned} \text{Then heart weight} &= \text{B.P.} \times \sqrt{H \times W} \times 0.02265 \\ &= 50 \times \sqrt{20.6 \times 7.55} \times 0.02265 \\ &= 14.10 \text{ grams} \end{aligned}$$

This is a remarkable computation since Holt gives the weight of the average heart at birth as 14.0 grams.

As a continuation of this study we will now develop the

theoretical values of heart weight, based on the value of $K_{(1)} = 0.02265$, for the few ages given by Holt and recorded in the following tabulation:

TABLE V

| Age | Height (Inches) | Weight (Pounds) | Blood pressure MacLaren and Others | Estimated Heart weight | Actual (Holt) |
|-------------|--------------------|--------------------|--|---------------------------|------------------|
| Birth | 20.6 | 7.55 | 50 m.m. | 14.1 gms. | 14.0 gms. |
| 1 year | 29.0 | 20.5 | 81(?) " | 44.7 " | 35.0 " |
| 2 years | 32.5 | 26.5 | 81 " | 53.8 " | 53.0 " |
| 3 " | 35.0 | 31.2 | 84(?) " | 62.9 " | 64.0 " |
| 7 " | 46.2 | 46.0 *1 | 88 " | 91.9 " | 80.0 " |
| 14 " | 61.0 | 91.0 *1 | 106 " | 178.6 " | 166.0 " |
| Adults(20?) | 67.0 | 134.0 *1 | 120 " | 257.4 " | 241.0 " |

(*1 Correction made for weight of clothing)

In Graph II we have developed a curve for theoretical weights of hearts for various ages, using the average heights and average body weights employed in developing Graph I, but in this instance, using our constant $K_{(1)} = 0.02265$. Because we have developed our constant $K_{(1)}$ on Mueller's data based on cadavers, we have made for adults a deduction in weight of 10 pounds for clothing, and in height, one inch for height of heels. We have inserted, for comparison, the heart weights calculated and presented in the foregoing table. The trends of the two curves are practically identical, and we believe that this study, though far from comprehensive through dearth of specific data on heart weights for corresponding body heights and weights, points definitely to a fundamental relationship between heart weight, blood pressure and body surface area. It suggests also the desirability of correlating all three factors in the study of cardiac roentgenograms and orthodiagrams.

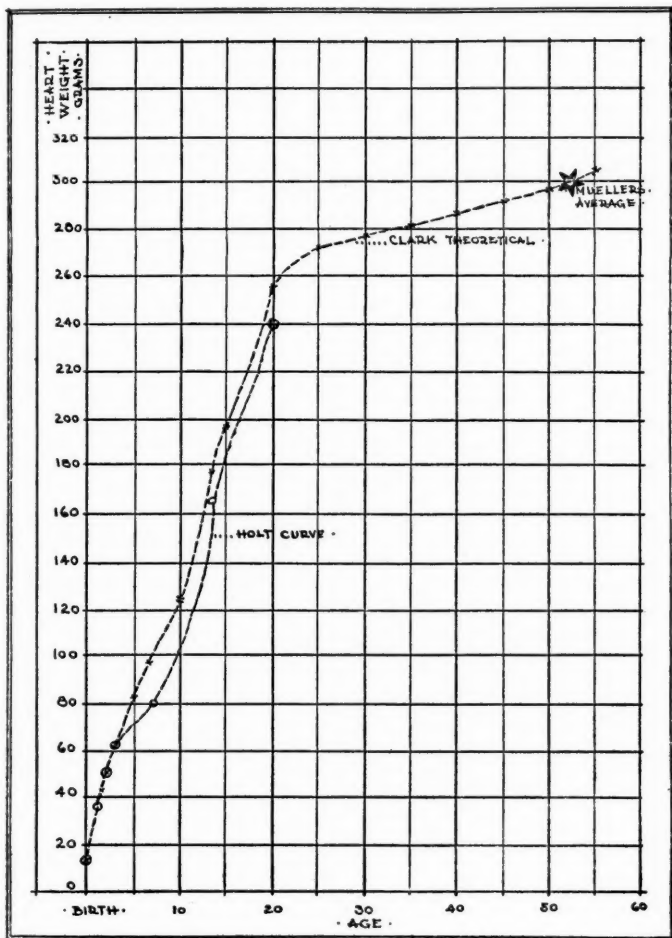
The foregoing studies suggest that:

- I. *The Weight Of The Heart, In Normal Subjects, Is Determined By The Surface Area Of The Body.*
(Note 3.)

This conception is in harmony with the observations of Rubner, DuBois and others that the Metabolic Rate varies with the skin area.

GRAPH II

Calculated heart weights compared to averages for infancy and childhood published by Holt. (See text and Table V)



It also is in harmony with the recent assertion of Grollman that the output of blood from the heart varies with the surface area of the body.

- II. *Height Of Blood Pressure Appears To Be Determined, In Normal Subjects, By The Ratio Which Exists Between The Force Or Weight Of The Heart And The Body Surface Area.*

Although pathological and nervous influences, chronic or even acute intoxications, muscular effort, etc., may cause temporary or permanent alteration of the heart force and consequently of the normal pressure, we may state that, fundamentally, the rise of pressure from birth to advanced age appears to follow a simple formula of physics.

- III. *The Importance Of Body Surface Area In The Physiology Of The Circulatory System Is Suggested, Since In Growth, It Appears To Determine Primarily The Size Of The Heart, And Secondarily The Height Of The Blood Pressure.*

We have thus far demonstrated that blood pressure may be studied either through its relationship to $\frac{\text{Heart Weight}}{\text{Surface Area}}$, or to $\frac{\text{Body Weight}}{\text{Surface Area}}$.

We will now present a third study based upon the relationship between blood pressure and the metabolic rate.

Since it was demonstrated that, during growth,

Blood pressure varies with $\frac{\text{Body Weight}}{\text{Surface Area}}$,

it occurred to the writer that the metabolic rate may vary in an inverse fashion; that is—possibly,

Metabolic rate varies with $\frac{\text{Surface Area}}{\text{Body Weight}}$.

To test this possibility, we developed a constant, $K_{(3)}$ —

$$K_{(3)} = \frac{\text{Surface Area}}{\text{Body Weight} \times \text{Metabolic Rate}}$$

By using the metabolic rate values of Boothby and Sanford and the Surface Area formula of DuBois, we ascertained that the numerical value of $K_{(3)} = 6.75$. (Note 3.) We then developed a table of theoretical values for metabolic rates for ages, birth to 55 years, and Graph III displays the result. We have also plotted a curve based on the table of Boothby and Sanford (ages 5 to 55 years). The theoretical values for ages 20 and over are in practical agreement, but again, as we did in our study of blood pressure and also of heart weight, we find a disagreement in childhood. We are led to believe that these deviations all arise from the same factor, that is, our surface area values for children, due probably to the fact that the average weights for children, as recorded in Holt's table, are not identical with the weights of children on whom the other studies were made. A slight change in weight or height would cause a perfect agreement between actual or estimated rate for any of the ages recorded in our study. (Note 5.)

That the deviations noted in our three foregoing studies are probably due to our surface area values, is indicated by the following study. If blood pressure varies with $\frac{\text{Body Weight}}{\text{Surface Area}}$,

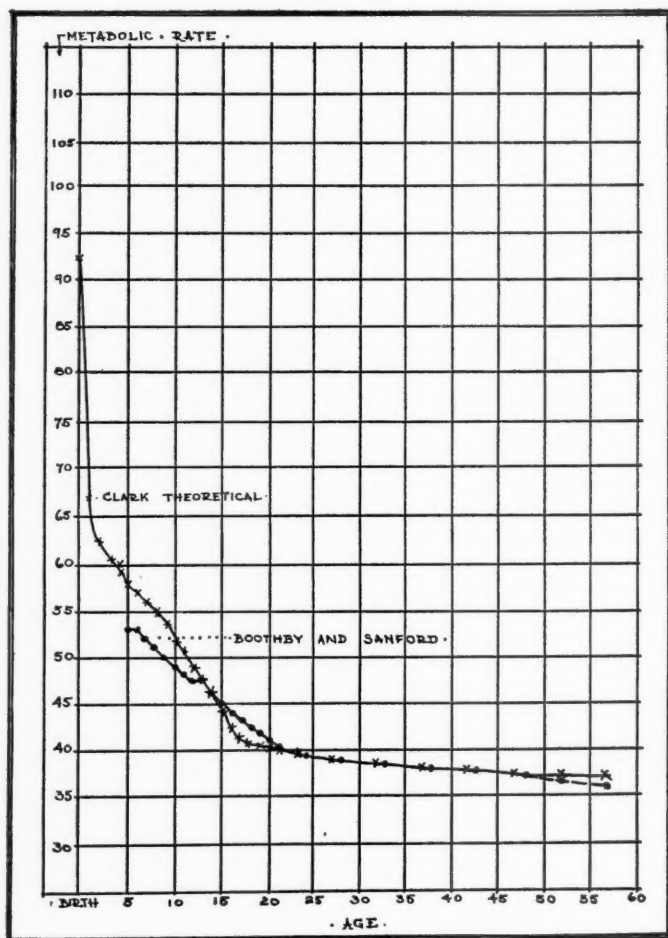
and metabolic rate varies with $\frac{\text{Surface Area}}{\text{Body Weight}}$, then it must follow

that as blood pressure rises the metabolic rate must fall. We should then ascertain whether the product of pressure and metabolic rate remains constant during growth. The following table demonstrates that this constant relationship does occur for the ages studied and the value of the constant—Blood Pressure \times Metabolic Rate—is approximately 4,810.

In Column A (Table VI) are given the blood pressure values, ages Birth to 2 years, taken from MacLaren's table; ages 3 to 15 years from Judson and Nicholson; ages 20 to 60 from Hunter. In Column B are given the metabolic rates according to Boothby and Sanford. In Column C are given the product of A and B. In Column D are given the estimated metabolic rates secured by dividing the constant 4,810 by the blood pressure for each age (Column A). Column E gives theoretical blood pressure values

GRAPH III

Calculated Metabolic Rates compared to averages of Boothby and Sanford.



Study of Blood Pressure

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based on constant 4,810 divided by metabolic rates of Boothby and Sanford (Column B). These values are strikingly similar to those given in Column A, which were based upon the study of many hundred thousand observations.

TABLE VI

| | A | B | C | D | E |
|---------|-----------|-----------|------------------|---------------------------------|---------------------------------|
| Age | B.P.—M.M. | Met. Rate | A × B | $\frac{4,810}{\text{B.P. (A)}}$ | $\frac{4,810}{\text{M.R. (B)}}$ |
| Birth | 50 | ? | | 96.1 | |
| 1 year | 80 | ? | | 60.0 | |
| 2 years | 81 | ? | | 59.4 | |
| 3 " | 91.8 | ? | | 52.4 | |
| 4 " | 91.6 | ? | | 52.5 | |
| 5 " | 91.3 | (53) | 4,839 | 52.7 | 90.8 |
| 6 " | 92.6 | 53 | 4,908 | 51.9 | 90.8 |
| 7 " | 94.0 | 52 | 4,888 | 51.2 | 92.5 |
| 8 " | 93.6 | 51 | 4,774 | 51.4 | 94.3 |
| 9 " | 94.3 | 50 | 4,715 | 51.0 | 96.0 |
| 10 " | 99.2 | 49 | 4,861 | 48.5 | 98.2 |
| 11 " | 97.1 | 48.5 | 4,710 | 49.5 | 99.2 |
| 12 " | 102.3 | 47.5 | 4,859 | 47.0 | 101.3 |
| 13 " | 103.6 | 47.0 | 4,869 | 46.4 | 102.3 |
| 14 " | 106.1 | 46.0 | 4,881 | 45.4 | 104.6 |
| 15 " | 105.6 | 45.0 | 4,752 | 45.5 | 107.0 |
| 20 " | 120 | 41.0 | 4,920 | 40.0 | 117.3 |
| 25 " | 122 | 39.5 | 4,819 | 39.4 | 121.8 |
| 30 " | 123 | 39.0 | 4,797 | 39.1 | 123.3 |
| 35 " | 124 | 38.5 | 4,774 | 38.8 | 124.9 |
| 40 " | 126 | 38.0 | 4,788 | 38.2 | 127.5 |
| 45 " | 128 | 37.5 | 4,800 | 37.6 | 128.3 |
| 50 " | 130 | 37.0 | 4,810 | 37.0 | 130.0 |
| 55 " | 132 | 36.0 | 4,752 | 36.4 | 133.3 |
| 60 " | 135 | 35.5 | 4,793 | 35.6 | 135.4 |
| | | | 20 <u>96,219</u> | | |
| | | | Average . . . | 4,810 | |

From this third study it appears justifiable to conclude that, during growth, and probably throughout life, insofar as healthy men of average height and weight for age are concerned,

I. The Height of Blood Pressure Varies Inversely With Metabolic Rate.

Furthermore, it appears that,

- II. *Although The Law Of Richet-Rubner States That The Caloric Requirement Is Determined By The Surface Area Of The Body, It Appears That The Body Weight Must Be Considered In The Development Of A Constant For Metabolic Rates For Man. (Note 6.)*

We may also suggest that,

- III. *The Height Of Blood Pressure Of An Individual May Well Be Compared, Not Only To The Average For Age, But Also To The Theoretical Average For Surface Area And Body Weight Ratio.*

It has been suggested that I give to those of our group working in the field of Röntgenology, my further suggestions relative to the value of build indices which may be of practical application to the study of the heart through the aid of the Röntgen ray.

We are concerned with three factors: First, heart diameters which are linear and therefore one-dimensional; second, the heart shadow which is a plane and therefore two-dimensional; third, heart volume or heart weight which is three-dimensional.

In a previous paper presented to this association we demonstrated that the body circumferences vary with the index $\frac{w}{h}$. The same index, and the index $\sqrt{\frac{w}{h}}$ are applicable to the study of body diameters. They are, also, I believe, equally serviceable in the study of heart diameters as demonstrated by the writer in a preliminary analysis of a small group of cases. (See Graph IV.) Furthermore, we have shown that blood pressure also varies in normal averages with $\frac{\text{Body weight}}{\text{Surface area}}$ or $\frac{w}{\sqrt{w h}}$.

Therefore

$$(\text{B.P.})^2 \propto \frac{w^2}{w h} \text{ and } \text{B.P.} \propto \sqrt{\frac{w}{h}}.$$

Blood pressure may be, therefore, considered one-dimensional.

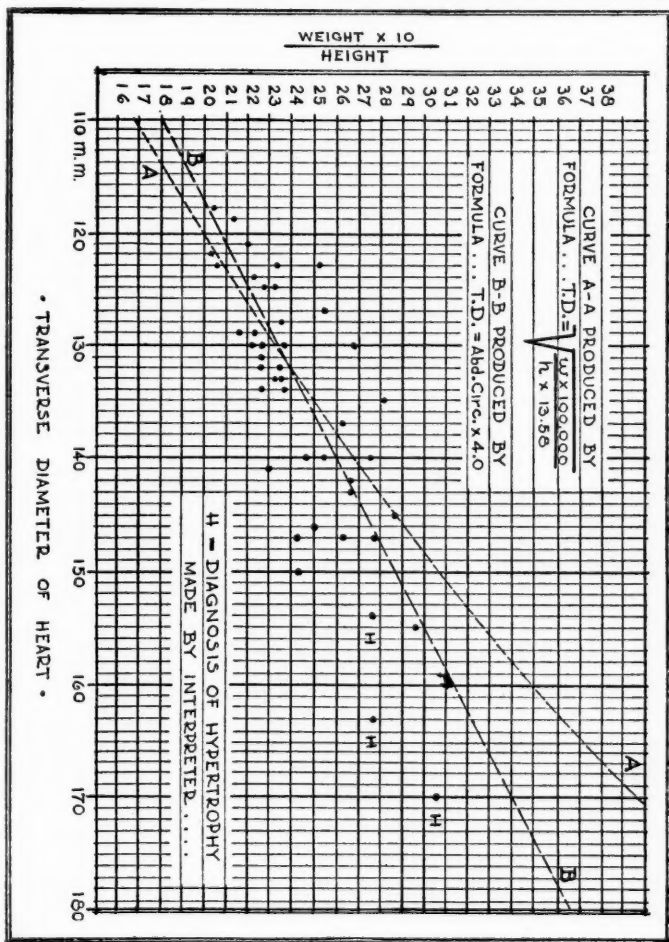
GRAPH IV

An Illustration of the Correlation Between T.D. of Heart and—

I. The Height-Weight Index $\frac{w \times 10}{h}$,

II. Abdominal Circumference—Based on Theoretical Circumferences Given in Author's Table of Chest and Abdominal Circumferences.

(A study of 43 X-ray Films Submitted with Insurance Examinations)



The heart shadow as above stated is two-dimensional. Furthermore, the surface area of the body is two-dimensional; since the body resembles a cylinder, we may, for the purpose of study, compare the surface areas of two human beings by the ratio which exists between the product of height and, for example, the abdominal girth, and use the index $(h \times c)$.

Translating the index $h \times c$ into terms of height and weight, then surface area may be studied, also, either with the index $\sqrt{w} h$ or by the DuBois index $h^{.725} w^{.425}$.

We have demonstrated in our paper that we may use the following constant for the study of heart weight:

$$K = \frac{\text{Heart Weight}}{\text{Surface Area} \times \text{Blood Pressure.}}$$

The denominator of this constant is three-dimensional, just as is the numerator, for we have shown that surface area is two-dimensional and we have also demonstrated that blood pressure may be considered as one-dimensional.

In our paper we have utilized constants, the numerical values of which have been developed from data on adults, and we have then derived curves based on average weight for height from birth to late adult ages. From a practical application to future studies, particularly because of the effect of underweight and overweight, I believe better results will be obtained in the study of any age group if we use indices and then plot curves. These curves may then be smoothed out by special mathematical methods. By referring to my original paper on chest and abdominal circumferences, it will be observed that the theoretical circumferences of chest and abdomen for each value of $\frac{w}{h}$ were developed by this method. My suggestion is, therefore, that it may be well to study heart diameters by the use of the index $\frac{w}{h}$ or $\sqrt{\frac{w}{h}}$, and area of heart silhouette with the index $\sqrt{w} h$. We have also noted a probable close correlation between the transverse diameter of the heart and the abdominal circumference, and in

Graph IV, $K = \frac{T D \text{ (m.m.'s)}}{\text{Abd. Circumference (inches)}}$.

The value of K as indicated by this study is approximately 4.0

and therefore $TD = c \times 4$ and the transverse diameter of the heart appears to be in millimeters approximately four times the abdominal girth in inches.

Another suggestion which we might reasonably make is that, since the area of the cardiac silhouette is two-dimensional, we may use, for our denominator, body height as the first, and blood pressure the second dimension. Therefore $\frac{\text{Area of Silhouette}}{\text{Height} \times \text{B.P.}}$ may be suggested as a possible constant.

TABLE VII
A
Test of $\frac{A}{H \times B. P.}$

| Age | Height | B.P.*1 | Cardiac Area**2 | $\frac{A}{H \times B. P.}$ | Estimated Cardiac Area $A = H \times B.P. \times K$ |
|--------|--------|--------|-----------------|----------------------------|--|
| 3 yrs. | 35.0 | 91.8 | 44.4 | 0.0138 | 43.4 |
| 4 " | 38.0 | 91.6 | 47.8 | 0.0137 | 47.0 |
| 5 " | 41.7 | 90.8 | 52.4 | 0.0139 | 50.9 |
| 6 " | 44.1 | 90.8 | 55.3 | 0.0138 | 54.0 |
| 8 " | 48.2 | 94.3 | 61.5 | 0.0135 | 61.4 |
| 10 " | 52.2 | 98.2 | 69.3 | 0.0135 | 69.2 |
| 12 " | 55.8 | 101.3 | 77.0 | 0.0136 | 76.3 |
| 15 " | 63.0 | 107.0 | 95.0 | 0.0141 | 91.0 |
| 18 " | 67.0 | 113.0 | 105.3 | 0.0139 | 103.7 |
| 20 " | 68.0 | 117.3 | 108.8 | 0.0136 | 107.7 |
| 25 " | 68.0 | 121.8 | 109.7 | 0.0132 | 111.8 |
| 30 " | 68.0 | 123.3 | 110.2 | 0.0131 | 113.2 |
| 35 " | 68.0 | 124.9 | 110.7 | 0.0130 | 114.9 |
| 40 " | 68.0 | 127.5 | 111.1 | 0.0128 | 117.0 |
| 45 " | 68.0 | 128.3 | 111.4 | 0.0128 | 117.8 |
| 50 " | 68.0 | 130.0 | 111.6 | 0.0126 | 119.3 |
| 55 " | 68.0 | 133.3 | 111.9 | 0.0124 | 122.4 |

$K = \text{Average} = 0.0135$

*1. Blood pressures taken from MacLaren (ages 3 and 4 years) and Column E, Table VI (ages 5-55).

*2. Cardiac areas from prediction tables of Eyster and the recent table for children by Hodges, Adams and Gordon. In Eyster's table no correction is made for age, though such correction is advised for T.D. of heart. This may account for the decreasing index after age 20.

From the various tests of the many indices which we have made, we are convinced that the height of blood pressure—being apparently, in measure, an index of the distance of the surface area from the heart center—is of the greatest importance in estimating heart size and should be included, when possible, in our indices and constants.

We would recommend, in addition to other investigations suggested in this paper, that we ascertain in future studies the degree of correlation which may exist between—

- I. *The transverse diameter of the heart and circumference of the abdomen. If such close correlation should be found, it could readily be explained through the fact that the abdominal girth more accurately reflects the amount of fat deposit than any other body circumference. It is recognized that the same factor influences the position of the heart.*
- II. *Area of cardiac silhouette, and product of blood pressure and height.*
- III. *Weight or volume of the heart and the product of blood pressure and body surface area.*

In conclusion, it should be stated that the writer has presented herewith certain studies of the relationship between systolic blood pressure and heart weight, body surface area and metabolic rate for infants, children and adults of average height and weight, through the aid of published data derived from many sources. The effect of underweight and overweight may materially modify the constants presented, and much further data must be obtained before such effect can be demonstrated. The paper is presented at this time with the thought that it should be of interest to others and may be of value in future studies of the circulatory system.

NOTE 1

For the calculation of Surface Area in square centimeters we find that—

$h^{.725} w^{.425} \times 71.84$ (DuBois) approximately equals $\sqrt{h.w.} \times 167$, and for the ages included in our study the calculations differ only slightly, e. g.,

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| | $h^{.735} w^{.485} \times 71.84$ | $\sqrt{h.w.} \times 167$ |
|----------|----------------------------------|--------------------------|
| Birth | 2,143 sq. cms. | 2,224 |
| 10 years | 10,568 " " | 10,571 |
| 17 " | 17,074 " " | 17,004 |
| 27 " | 18,112 " " | 18,126 |
| 37 " | 18,463 " " | 18,545 |
| 47 " | 18,662 " " | 18,779 |
| 57 " | 18,727 " " | 18,887 |

It should be noted that—

Our constants $K_{(1)}$ and $K_{(2)}$ are based on the index $\sqrt{h.w.}$ and not the square centimeters of skin surface.

NOTE 2

Holt, in his table of heart weights, has given the ratios of heart to body weights, from which ratios we can approximate the body weight. The following table gives the value of $K_{(1)}$ for each age based on the actual heart and body weights of the subjects studied. Heights are assumed to be identical with those of living children, and blood pressures are taken or estimated from MacLaren's table:

| Age | Height | Weight | B.P. (m.m.) | Heart (Gms.) | $K_{(1)}$ |
|----------------|--------|-----------|----------------|-----------------|-----------|
| Birth | 20.6" | 6.93 lbs. | 50 | 14 | 0.0234 |
| 1 year | 29.0" | 17.3 " | 81 | 35 | 0.0193 |
| 2 years | 32.5" | 26.2 " | 81 | 53 | 0.0224 |
| 3 " | 35.0" | 31.7 " | 84(?) | 64 | 0.0229 |
| 7 " | 46.2" | 49.3 " | 88(?) | 80 | 0.0191 |
| 14 " | 61.0" | 81.0 " | 106(?) | 166 | 0.0224 |
| Adult (20?) | 67.0" | 119.8 " | 120 | 241 | 0.0224 |
| Mueller's (50) | 67.0" | 155.0 " | 130 | 300 | 0.0226 |

It is apparent that slight variations in the values of height, weight, and particularly blood pressure will materially affect the value of a constant. Considering that fact the above values for $K_{(1)}$ are remarkably consistent.

NOTE 3

At least two major factors must be considered in estimating the work of the heart—first, the surface area which must be supplied with blood, and second, the distance which the required volume of blood must be projected by the heart. It is obvious that the force of the heart must increase as the distance of the surface area from the heart increases. The following table gives the approximate number of grams of heart weight for each 100 sq. cm. of surface area for the ages for which we have data on heart weight. (See Note 2—height and weight converted to metric measurements.)

| Age | Height (Cms.) | Weight (Kgms.) | Surface Area (Sq. Cms.) *1 | Heart Weight (Grams) | Grams Heart Weight for each 100 Sq. Cms. Surface Area |
|-------------------|------------------|-------------------|-------------------------------|-------------------------|--|
| Birth | 52.0 | 3.15 | 2,053 | 14 | 0.682 |
| 1 year | 73.7 | 7.86 | 3,897 | 35 | 0.898 |
| 2 years | 82.5 | 11.91 | 5,048 | 53 | 1.050 |
| 3 " | 88.8 | 14.41 | 5,772 | 64 | 1.109 |
| 7 " | 117.0 | 22.41 | 8,506 | 80 | 0.940 |
| 14 " | 154.5 | 36.82 | 12,850 | 166 | 1.292 |
| 20(?) " | 170.0 | 54.45 | 16,265 | 241 | 1.482 |
| 50 " (Mueller) | 170.0 | 70.45 | 18,149 | 300 | 1.653 |

*1 DuBois formula.

It is probable that the increase in heart weight noted in the last column is, for the most part, due to the increase in distance of skin surface from the heart center.

NOTE 4

CONSTANT FOR METABOLIC RATE

We have based our constant $K_{(3)}$ on the following metabolic rates from the table of Boothby and Sanford.

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| Age | Height | Weight | Surface Area *1 | Met. Rate |
|-------------------------|-----------|---------|-----------------|-----------|
| 20 years | 172.8 cm. | 65.45 K | 17,795 | 41.0 |
| 21 " | 172.8 " | 65.91 K | 17,850 | 40.5 |
| 23 " | 172.8 " | 66.72 K | 17,942 | 40.0 |
| 27 " | 172.8 " | 68.18 K | 18,112 | 39.5 |
| 32 " | 172.8 " | 70.00 K | 18,313 | 39.0 |
| 37 " | 172.8 " | 71.36 K | 18,463 | 38.5 |
| 42 " | 172.8 " | 72.27 K | 18,564 | 38.0 |
| 47 " | 172.8 " | 73.18 K | 18,662 | 37.5 |
| 52 " | 172.8 " | 73.64 K | 18,711 | 37.0 |
| 57 " | 172.8 " | 74.09 K | 18,727 | 36.0 |
| | | 700.80 | 183,139 | 387.0 |
| Average—172.8 Cm. . . . | | 70.08 | 18,314 | 38.7 |

$$K = \frac{18,314}{70.08 \times 38.7} = 6.75$$

*1 Surface Area in sq. cm. calculated by DuBois formula $SA = h^{.725} \times w^{.425} \times 71.84$. No correction made for clothing or height of heels.

NOTE 5

It is improbable that two investigators could ever agree exactly upon the metabolic rates for all ages, since it would necessitate that the average height and weight for each age studied by the two investigators should agree. The following table shows the close agreement of the theoretical rates of the writer to the table of Boothby and Sanford, and that of DuBois:

| Age | Boothby and Sanford | Clark Theoretical | DuBois |
|----------|---------------------|-------------------|--------|
| 15 years | 45.0 | 44.3 | 46.0 |
| 17 " | 43.5 | 41.5 | 43.0 |
| 19 " | 42.0 | 40.6 | 41.0 |
| 27 " | 39.5 | 39.4 | 39.5 |
| 37 " | 38.5 | 38.3 | 39.5 |
| 47 " | 37.5 | 37.8 | 38.5 |
| 57 " | 36.0 | 37.4 | 37.5 |

NOTE 6

That the constant $K_3 = \frac{S}{W \times M}$ is applicable to the study of the Metabolic Rate for man appears probable, because:

First—The curve based on the Estimated Metabolic Rates by the use of K_3 closely parallels that based on the averages determined in the laboratory and published by Boothby and Sanford.

Second—The products of Blood Pressure and Metabolic Rates for ages 5 to 60 years, we have found to be constant, just as the product of K_2 and K_3 is a constant;

$$(i. e., \frac{W}{S \times B.P.} \times \frac{S}{W \times M} = \frac{1}{B.P. \times M})$$

Since the Metabolic Rate signifies the number of calories of heat developed per hour per square meter of skin surface, we may then state that:

$$M = \frac{C}{S}$$

in which formula M = Metabolic Rate, S = Square meters of skin surface and C = Calories of heat per hour. Therefore:

$$\begin{aligned} K_3 &= \frac{S}{W \times M} \\ &= \frac{S}{W \times \frac{C}{S}} = \frac{S \times S}{W \times C} \end{aligned}$$

It would appear, as we pass from youth to age, that the first determinant of the metabolic rate is body weight; the body weight determines the surface area; and the surface area determines the number of Calories of heat developed.

The conclusion of J. J. R. Macleod, as quoted by Gradwohl and Blavais, is as follows:

"The determining factor of energy output is undoubtedly the general condition of bodily nutrition—the active mass of protoplasm of the body (Benedict). That there is a relationship between the body surface and metabolism is undoubted, but the relationship is not a causal one."

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It appears likely, from the study here presented, that both the quantity of active protoplasmic mass and the surface area are equally important factors in determining the constant for the metabolic rates during growth and also for healthy adults. The numerical value of the constant for man does not appear, however, to be identical with the constants for lower animals (e. g., horse, rabbit and mouse).

DR. COOK—I have read and re-read Dr. Clark's paper with the greatest interest and profit. An original piece of work which reveals truth and clarifies our conceptions is always stimulating and refreshing, and particularly so when many of our papers must of necessity be summaries of existing clinical knowledge or analyses of statistical evidence. It is peculiarly felicitous that this original contribution of Dr. Clark's should give us a better insight into the cause and significance of the most important single physical sign which is developed in an insurance examination—blood pressure. I think no one can read Dr. Clark's paper without deep appreciation of the conclusive way in which he directs our thoughts, and will, I trust, lead our further investigation into the relation in pathological and impaired conditions which may exist between blood pressure, cardiac hypertrophy, and overweight. It has long been recognized both clinically and statistically that such a relationship existed, but Dr. Clark gives us a new significance and renewed interest to further studies along these lines.

The late Dr. Gordon Wilson at the 1931 meeting of this Association had an excellent paper, which emphasized the importance of recognizing cardiac enlargement in life insurance examinations. In a discussion of this paper, I stated, "I wish Dr. Wilson had laid more stress on blood pressure as evidence of cardiac disease, because in an insurance examination it assumes the first importance. Hypertension is our best single underwriting evidence of cardiac hypertrophy, of far greater significance than percussion or location of apex beat. It is almost safe to say as an underwriting dictum, that if we have hypertension we have hypertrophy, and with normal tension we do not have hy-

per trophy in the absence of valvular defect or failing myocardium". Dr. Clark's paper takes us a long step forward in a better understanding of the relationship of these three sinister factors in cardiovascular disease—overweight, cardiac hypertrophy, and hypertension.

The outstanding problem today in medical selection is the early detection of the chronic degenerative forms of cardiovascular disease, and the triad of overweight, cardiac hypertrophy, and hypertension are our most obvious danger signals.

There has undoubtedly been too lenient an attitude taken of late towards overweight at the older ages, and revision of some height and weight tables is being made to bring ratings more in line with experience.

The detection of cardiac hypertrophy is beyond the ability, time, and technique of the ordinary insurance examination, and it but rarely appears on an insurance blank except in connection with valvular insufficiencies, and the large risk where an X-ray is required.

Hypertension is more frequently associated with beginning cardiovascular disease than any other discoverable sign, and it is easily estimated by any physician worthy of the name or an appointment as examiner. It is certainly an anachronism today to give consideration to examiners' ability to take blood pressures, when trained nurses can be easily taught to take accurate readings. All the operative and postoperative blood pressures at St. Mary's Hospital in Rochester are taken by nurses.

While our examiners are undoubtedly capable of giving us correct blood pressures today we are unfortunately not taking full advantage of this pre-eminently important sign, for two reasons:

First, because examiners frequently give the minimum reading obtainable, and often even shade this 10 or more mm. We can do much to correct this dangerous tendency by continued efforts to educate them as to our wishes, and by cautioning them against some fallacious clinical opinions, i.e., that hypertension is not of great significance, that it is temporarily due to smoking, exercise, an abscessed tooth, nervousness, etc.

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The second and more important reason why we are not preventing more of the early and serious losses from cardiovascular disease is that we are not acting on the lesser degrees of hypertension with the severity they deserve. We have not yet learned to rate hypertension as statistical experience indicates is necessary nor in accordance with the best posted clinical opinion which is expressed by Alvarez and Stanley after an examination of 6,000 individuals, "A pressure of 115 mm. Hg. is just as normal, and a pressure of 140 mm. Hg. is just as abnormal in an old man as in a young one". Dr. Hunter cautioned us many years ago against mistaking "average" for "normal" in overweight and hypertension.

Many clinicians are entirely too casual in their estimation of the effects of moderate hypertension on the integrity and longevity of the cardiovascular system. Some have hypertension themselves and are loath to admit its significance, but with the majority it is due to the lack of sufficient experience and ignorance of statistical evidence. The same type of clinician who disregards the significance of heart murmurs following the optimism of Sir James Mackenzie and Dr. Richard C. Cabot is equally optimistic about hypertension. As a therapeutic reassurance to the hypertensive patient who is so often nervous, this is good clinical practice, but it becomes disastrous when translated into life underwriting. A group of entrants with 140+ mm. Hg. of systolic blood pressure can in my opinion never be successfully selected for a standard mortality. There are too many death claims from agina, coronary sclerosis, apoplexy, and other forms of cardiovascular disease being paid by all American companies today where the applicant five, ten, or fifteen years ago showed 136 mm. Hg. to 150 mm. Hg. systolic pressure.

Dr. Hunter stated this morning in his paper that the blood pressure ratings of the M.I.R. were made as low as they are because of the clinical opinions expressed by the committee, and against definite statistical experience. In one case the rating is +30, where the actual experience was +80. I believe more extended experience will prove the correctness of the statistical experience and that clinical opinion was wrong, and that com-

panies which follow the M.I.R. on blood pressure will have a costly experience on applicants, especially over 40, with systolic pressures of 140 and over. A young girl or a country boy may have a rise in systolic pressure at the beginning of an examination, which is to them an unusual and an alarming experience. But the man of affairs of 50 who is so nervous at a medical examination that it sends his blood pressure up is either fearful that a known impairment will be discovered, or is in such an unstable nervous condition that some underlying organic condition may be suspected, or that his "functional" instability will lead to organic disease, and an established hypertension.

I again want to thank Dr. Clark for his inspiring contribution to our knowledge of blood pressure and its relation to weight and heart size.

DR. LIVINGSTON—May I point out that Dr. Clark carefully gives as the title of his paper, "*A Theoretical Study of Blood Pressure and Its Relation to Heart Size, Body Surface Area and Metabolic Rate*". The formulæ are developed in a logical manner, supporting evidence is given in detail and the proof is cleverly presented; at first sight it appears perfect, but my common sense judgment rebels against the omission of the age factor, for I believe age should be considered when we are dealing with average readings. One may question the substitution of body surface area for blood vascular area. With a constant height an increase in weight results in increase of body surface area, but I am not prepared to admit a similar and proportionate increase in blood vascular area. The increase in weight is largely, if not entirely, fat and water, and fat has a poorer blood supply than vital organs and muscle. One may also question the substitution of heart weight for heart force, and the substitution *particularly* of body weight for heart weight. However, with these substitutions, and bearing in mind we are dealing with a theoretical study, I admit the formulæ and conclusions are sufficiently striking to warrant further study.

I am inclined to give a considerable credit to endocrine balance for the relation of blood pressure to heart size, body surface

area and the metabolic rate, particularly if we consider a central average group only, as Dr. Clark has done. I admit this may be putting in another way what Dr. Clark has apparently proven. For ages up to 18 years we have a gradual increase in average height and weight, the result of growth, which we may well consider an endocrine function, and we would expect a definite relationship of height, weight, body surface area, metabolic rate, heart size and blood pressure. For ages 18 to 25 we may presume there is an endocrine function which results in gain in weight, with little or no increase in height, but gain in weight after 25 years I have always questioned as a normal function. Dr. Clark developed his constant K_2 from average values for adults, and it is remarkable that this constant can be accurately applied for average values for children; thus it appears to me that increase in weight as shown by averages after 25 years of age may well be a normal function of the endocrines. Small degrees of overweight and underweight can hardly be considered examples of endocrine imbalance, and the great majority of cases of even marked overweight and underweight can at worst be considered only as a physiological endocrine imbalance. I believe that after 20 years the age factor becomes of importance from an insurance and clinical viewpoint.

If one plots contour lines with build as ordinates and age as abscissæ, from the Joint Committee report on blood pressure 1925 Table 7 all heights, and then computes the values by Clark's formula for 5' 8", we find that both sets of lines cross at or very near the average weight line, but then diverge. As we would judge from the 1925 investigation and Clark's formula, the increase of blood pressure indicated by the slope of the surface represented by the contour lines is mostly a function of build in Clark's lines but mostly a function of age in the lines from the 1925 investigation.

From the 1925 investigation height is not an important factor except as it must be taken into consideration in determining build groups. The increase shown is only 5 m.m. from 5' to 6' 3", all ages, and the increase becomes steadily less rapid as the height increases. Clark's formula gives results agreeing with Table 12

of the 1925 investigation if one adheres to average build, but with overweight and underweight discrepancies occur. In addition, the increase with height becomes steadily more repaid as the height increases, as shown in the following table:

Age 40—All Average Weight for Height

| Height | Weight | Blood Pressure by Clark's Formula |
|--------|----------|--------------------------------------|
| 5' 0" | 131 lbs. | 122.3 m.m. |
| 5' 4" | 141 " | 122.9 " |
| 5' 8" | 158 " | 126.2 " |
| 6' 0" | 180 " | 130.9 " |
| 6' 4" | 206 " | 136.3 " |

You will note that Clark's formula shows a difference of 14 m.m. from 5' to 6' 4", which is much in excess of that shown in the 1925 investigation. If we construct a table for age 35 and blood pressure 124 m.m. we find—

| Height | Normal Weight | Weight for same B.P. by Clark (124 m.m.) | Percentage of Normal Weight |
|--------|------------------|--|--------------------------------|
| 5' 0" | 128 lbs. | 137 lbs. | 7% overweight |
| 5' 4" | 138 " | 146 " | 6% " |
| 5' 8" | 155 " | 155 " | Normal |
| 6' 0" | 176 " | 164 " | 7% underweight |
| 6' 4" | 201 " | 173 " | 14% " |

You will thus see that short men must be overweight, and tall men must be underweight to give readings which approximate the average for age and normal build.

In Volume 9 of the Proceedings of the Association, Dr. Symonds' table shows about an equal spread in build groups as there is in age, viz., 11 m.m., and you will note that he gives 123 m.m. for average build ages 15 to 19. This appears somewhat high to me, and in fact is almost identical with the average for all ages and all builds as shown by the 1925 investigation, viz., 123.7 m.m. It thus appears that Dr. Symonds' table may under-estimate the importance of age according to more recent evidence and opinion of the true average at the younger ages. Using Clark's formula we can construct the following table for age 40, 5' 8"—

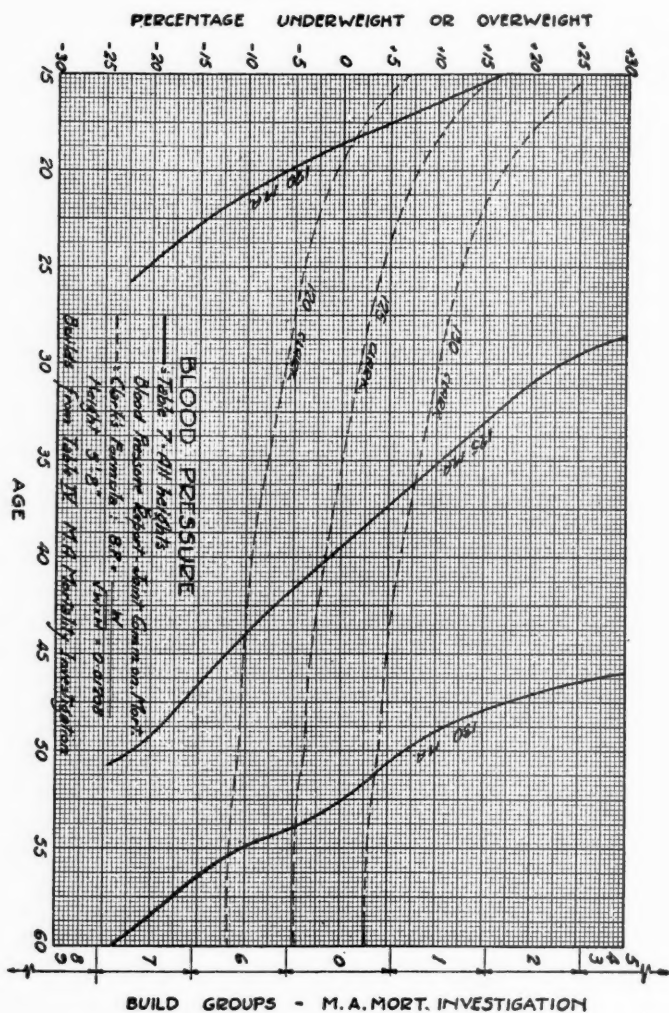
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| Build | Weight (in lbs.) | Blood Pressure |
|-----------|---------------------|-------------------|
| 30% u/wt. | 111 | 105.8 m.m. |
| 20% " | 126 | 112.7 " |
| 10% " | 142 | 119.6 " |
| Average | 158 | 126.2 " |
| 10% o/wt. | 174 | 132.4 " |
| 20% " | 190 | 138.4 " |
| 30% " | 205 | 143.7 " |
| 40% " | 221 | 149.2 " |

This gives us a spread of 43.4 m.m. for build from 30% underweight to 40% overweight, which is very much greater than shown by Symonds or the 1925 investigation. Similarly, it is out of line with the earlier work of Fisher and Hunter and Rogers. The 1925 investigation shows a spread for build of 10 m.m. and about one-fourth represents the change in age without reference to build.

In the following table are shown similar blood pressures by Clark's formula—all builds are average for age:

| Height | Weight | Blood Pressure | Average Build at Age |
|--------|----------|-------------------|-------------------------|
| 5' 0" | 128 lbs. | 121 m.m. | 35 |
| 5' 4" | 137 " | 121 " | 31 |
| 5' 8" | 145 " | 121 " | 21 |
| 6' 0" | 154 " | 121 " | 16 |
| 5' 0" | 131 " | 122 " | 40 |
| 5' 4" | 140 " | 122 " | 37 |
| 5' 8" | 148 " | 122 " | 24 |
| 6' 0" | 158 " | 122 " | 18 |
| 5' 0" | 133 " | 123 " | 45 |
| 5' 8" | 151 " | 123 " | 28 |
| 6' 0" | 160 " | 123 " | 19 |
| 6' 2" | 164 " | 123 " | 16 |
| 5' 0" | 135 " | 124 " | 55 |
| 5' 8" | 153 " | 124 " | 31 |
| 6' 0" | 162 " | 124 " | 21 |
| 6' 3" | 169 " | 124 " | 16 |
| 5' 4" | 145 " | 125 " | 55 |
| 5' 6" | 150 " | 125 " | 42 |
| 5' 8" | 154 " | 125 " | 33 |
| 6' 0" | 163 " | 125 " | 22 |
| 5' 8" | 163 " | 128 " | 55 |
| 5' 10" | 168 " | 128 " | 40 |
| 6' 0" | 173 " | 128 " | 31 |
| 6' 2" | 177 " | 128 " | 24 |



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This table clearly shows the effect of eliminating the age factor in the formula.

If Clark's formula is true that blood pressure varies with heart weight divided by body surface area then we would judge from Mueller's group of normal heart weights that the blood pressure would be lower for overweight than for average weight, and the blood pressure for underweight would be higher than for average weight. The formula as applied, however, gives contrary figures.

Age is a factor in heart size formula, and accordingly I feel should be included in any blood pressure formula. Dr. Clark I believe agrees, for his third conclusion is, "The height of blood pressure of an individual may well be compared not only to the average for age but also to the theoretical average for surface area and body weight ratio".

One must appreciate that Dr. Clark's paper is a preliminary study of a theoretical subject. Undoubtedly he had great difficulty in obtaining sufficient data with which to work. I feel we still have much to learn regarding blood pressure but I hope that further study along the line that Dr. Clark has suggested may give results.

DR. SCADDING—Dr. Clark, have you some additional remarks to make?

DR. CLARK—I wish to state that I, personally, found that my constant $K_{(2)}$ cannot be used in estimating the blood pressure for underweights and overweights at a given age. This is due, I believe, to the fact that the ratio of heart weight to body weight decreases as we pass from underweight to overweight. This is demonstrated by Mueller's table. However, I was justified in making a study of the blood pressure from birth to late adult age with constant $K_{(2)}$ in which I have substituted body weight for heart weight in constant $K_{(1)}$. This is possible since the ratio of heart weight to body weight from birth to late adult age for males of average height and weight for age is quite uniform—about 1 to 225 according to Holt.

It appears probable that we may be able to develop with constant $K_{(1)}$ an efficient prediction table for heart weight or

heart volume. This, I believe, because of a preliminary study which I have made with Mueller's averages including for each weight a probable height.

I wish to emphasize once more that the effect of underweight or overweight on any one or all of the constants proposed must be studied with more data than I have been able to find in the literature. There will doubtless be many divergent opinions expressed on the propositions which I have formulated, but it appeared to me well worth while to present the study to our association because of the original observations incorporated.

DR. SCADDING—Gentlemen, we have now concluded what I feel has been a most interesting and instructive program, and another of our annual meetings is drawing rapidly to a close. I recall that a year ago, when you so signally honored me by selecting me as your presiding officer, I launched out on my duties with many inward qualms and misgivings because of the noteworthy accomplishments of my predecessors. I failed to entirely realize at the time that, through the generous and untiring cooperation of the various committees, individual members and guest speakers, we would again be able to present a program which would materially advance the science of Life Insurance Medicine. And so, this hour to me means more than the conclusion of a successful program; it means the completion of a year replete with new friendships and the renewal of the old, and a year overflowing with the kindly wisdom and the friendly cooperation of those with whom I have been associated. The quality of loyalty and measure of support which instantly surrounded me after my election last year have carried me clean over many hurdles that at first seemed insurmountable. While the job of the chief officer of this Association is no sinecure, let no prospective president be dismayed. To be sure, he will have enough work to keep him out of mischief for a year but he will find an army of highly trained soldiers ready and willing to serve at his command. I speak as one who has every reason to know that this is the case, and whose gratitude is as profound as it is sincere.

Dr. Scadding's Closing Address 257

On behalf of the Association, let me offer our warmest thanks to all our contributors, both guests and members.

During the forthcoming twelve months, many new problems will be met and solved, new ideas will be conceived and developed, new friendships will be made and cherished, and new strands will be added to those ties which already bind our two great nations so closely together. This Association forms a necessary part of the whole of Life Insurance, a business which is imbued with the highest of humanitarian principles. I think it safe to say that we have never failed to play our small but important part in it.

While feeling the enormous power of the forces of Depression, Life Insurance has been the chief line of Defense, beyond which "They shall not pass". Its strength lies in its character, and who can doubt that character is the main factor in economic recovery. In this country we are not under the jurisdiction of the National Recovery Act. It might be better for us if we were. Referring to the real significance of the Act, one of your bishops has said that if it is only concerned with higher wages, old fashioned prosperity, mass production, and mass thinking, it is not worth fighting for; but if there is something more glorious to it—if it be true that America is at last groping for its soul—if we can see in it the glimmering light of the Brotherhood of Men—if we can see in it the radiance of the Fatherhood of God, then it is worth asking the best gift of God's grace that we may stand faithful and serve well. America is being asked to "come up higher". Where she goes, we usually follow.

It is now my proud privilege to introduce your presiding officer for the forthcoming year, Dr. Robert A. Fraser, Medical Director of the New York Life Insurance Company. May I bespeak for him the same degree of cooperation and loyalty you have shown to me. Nothing finer could happen to him. Dr. Fraser, will you take the chair?

DR. FRASER—After the admirable exhibition of inspirational literary effort of tongue and pen to which we have been treated,

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I am going to limit myself to a sincere appreciation of the signal honor accorded to my Company and myself. I thank you.

Dr. Weisse has a formal motion to present.

DR. WEISSE—On behalf of the members of this Association, I wish to present a motion of appreciation and thanks to Dr. Scadding and the members of the Canada Life Assurance Company who have furnished this Association with one of the finest meetings we have ever held, not only from the viewpoint of the quality and the amount of material furnished at the regular sessions but also from that of the royal entertainment furnished. The personal charm and friendly courtesy of Dr. Scadding and of his able lieutenants added that imponderable factor which makes such a meeting perfect.

The motion was seconded and received with hearty applause.

ADJOURNMENT

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| | |
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| Cecil C. Birchard, M. D. | Sun Life Assurance, Mon- treal, Que. |
| Arthur B. Bisbee, M. D. | National Life, Montpelier, Vt. |
| Wilton F. Blackford, M. D. | Commonwealth Life, Louis- ville, Ky. |
| David N. Blakely, M. D. | New England Mutual, Boston, Mass. |
| William Bolt, M. D. | New York Life, New York City. |
| Earl C. Bonnett, M. D. | Metropolitan Life, New York City. |
| J. Thornley Bowman, M. D. | London Life, London, Ont. |
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| Frederick G. Brathwaite, M. D. | Equitable Life Assurance, New York City. |
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| Willard B. Carpenter, M. D. | Columbus Mutual, Columbus, Ohio. |

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| Charles L. Christiernin, M. D. | Metropolitan Life, New York City. |
| Charles P. Clark, M. D. | Mutual Benefit, Newark, N. J. |
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| Henry W. Cook, M. D. | Northwestern National, Minneapolis, Minn. |
| Parker M. Cort, M. D. | Aetna Life, Hartford, Conn. |
| Donald B. Cragin, M. D. | Aetna Life, Hartford, Conn. |
| Hugh W. Crawford, M. D. | Connecticut General, Hartford, Conn. |
| Hamilton C. Cruikshank, M. D. | Manufacturers Life, Toronto, Ont. |
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| Robert M. Daley, M. D. | Equitable Life Assurance, New York City. |
| Joe E. Daniel, M. D. | Great Southern, Houston, Texas. |
| Harold D. Delamere, M. D. | Crown Life, Toronto, Ont. |
| Peter G. Denker, M. D. | Equitable Life Assurance, New York City. |
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| Edwin G. Dewis, M. D. | Prudential, Newark, N. J. |
| Henry K. Dillard, M. D. | Penn Mutual, Philadelphia, Pa. |
| Edward S. Dillon, M. D. | Penn Mutual, Philadelphia, Pa. |
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| William W. Dinsmore, M. D. | Travelers, Hartford, Conn. |
| Percy G. Drake, M. D. | Travelers, Hartford, Conn. |

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| Harold M. Frost, M. D. | New England Mutual, Boston, Mass. |
| Homer Gage, M. D. | State Mutual, Worcester, Mass. |
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| Robert A. Goodell, M. D. | Phoenix Mutual, Hartford, Conn. |
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| John K. Gordon, M. D. | Sun Life, Montreal, Que. |
| Angus Graham, M. D. | London Life, London, Ont. |
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| Robert J. Graves, M. D. | United Life & Accident, Con- cord, N. H. |
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| Llewellyn Hall, M. D. | Phoenix Mutual, Hartford, Conn. |
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| William J. Hammer, M. D. | New York Life, New York City. |
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| Walter C. Hausheer, M. D. | Prudential, Newark, N. J. |
| William D. Heaton, M. D. | New York Life, New York City. |
| Charles A. Heiken, M. D. | Home Life, Philadelphia, Pa. |
| Ernest M. Henderson, M. D. | Confederation Life, Toronto, Ont. |

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| William L. Hilliard, M. D. | Ontario Equitable, Waterloo, Ont. |
| Daniel W. Hoare, M. D. | Penn Mutual, Philadelphia, Pa. |
| William W. Hobson, M. D. | Reliance Life, Pittsburgh, Pa. |
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| Chauncey O. Hollinger, M. D. | Prudential, Newark, N. J. |
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| Washington C. Huyler, M. D. | Mutual Life, New York City. |
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| Carleton B. McCulloch, M. D. | State Life, Indianapolis, Ind. |
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| Frederick W. McSorley, M. D. | Equitable Life Assurance, New York City. |

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| Samuel W. Means, M. D. | Metropolitan Life, New York City. |
| William H. Miller, M. D. | Equitable Life Assurance, New York City. |
| W. Lindsay Miller, M. D. | Equitable Life Assurance, New York City. |
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| Robert C. Montgomery, M. D. | Manufacturers Life, Toronto, Ont. |
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| Alvin E. Murphy, M. D. | Prudential, Newark, N. J. |
| Charles T. Necker, M. D. | Dominion Life, Waterloo, Ont. |
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| James H. North, M. D. | New York Life, New York City. |

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| Charles B. Piper, M. D. | Connecticut Mutual, Hartford, Conn. |
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| William W. Quinlan, M. D. | Mutual Life, New York City. |
| Paul V. Reinartz, M. D. | Prudential, Newark, N. J. |
| Walter A. Reiter, M. D. | Mutual Benefit, Newark, N. J. |
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| Frank P. Righter, M. D. | Atlantic Life, Richmond, Va. |
| J. A. Roberts, M. D. | Canada Life, Toronto, Ont. |
| A. J. Robinson, M. D. | Connecticut General, Hart- ford, Conn. |
| Thomas H. Rockwell, M. D. | Equitable Life Assurance, New York City. |

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| Fred W. Rolph, M. D. | Confederation Life, Toronto, Ont. |
| Gordon Ross, M. D. | Massachusetts Mutual, Springfield, Mass. |
| Robert L. Rowley, M. D. | Phoenix Mutual, Hartford, Conn. |
| Charles L. Rudasill, M. D. | Life Insurance Company of Virginia, Richmond, Va. |
| Samuel C. Rumford, M. D. | Continental American, Wilmington, Del. |
| Eugene F. Russell, M. D. | Mutual Life, New York City. |
| H. Crawford Scadding, M. D. | Canada Life, Toronto, Ont. |
| C. E. Schilling, M. D. | Ohio State, Columbus, Ohio. |
| Samuel B. Scholz, Jr., M. D. | Penn Mutual, Philadelphia, Pa. |
| Albert Seaton, M. D. | American Central, Indianapolis, Ind. |
| George H. Shaw, M. D. | Travelers, Hartford, Conn. |
| Joyce T. Sheridan, M. D. | Fidelity Mutual Life, Philadelphia, Pa. |
| Arthur L. Sherrill, M. D. | Equitable Life Assurance, New York City. |
| Daniel M. Shewbrooks, M. D. | Penn Mutual, Philadelphia, Pa. |
| Ross L. Shields, M. D. | Mutual Life, Waterloo, Ont. |
| Ralph R. Simmons, M. D. | Equitable Life, Des Moines, Iowa. |
| Joseph L. Siner, M. D. | Fidelity Mutual, Philadelphia, Pa. |
| Donald W. Skeel, M. D. | Occidental Life, Los Angeles, Calif. |
| DeWitt Smith, M. D. | Southwestern Life, Dallas, Texas. |

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| James W. Smith, M. D. | American Central, Indianapolis, Ind. |
| Malcolm K. Smith, M. D. | Prudential, Newark, N. J. |
| William B. Smith, M. D. | Connecticut Mutual, Hartford, Conn. |
| Morton Snow, M. D. | Massachusetts Mutual, Springfield, Mass. |
| Marion Souchon, M. D. | Pan-American Life, New Orleans, La. |
| Howard B. Speer, M. D. | Metropolitan Life, New York City. |
| Henry F. Starr, M. D. | Pilot Life, Greensboro, N. C. |
| John B. Steele, M. D. | Volunteer State, Chattanooga, Tenn. |
| David F. Steuart, M. D. | Mutual Benefit, Newark, N. J. |
| Hector M. Stevenson, M. D. | Aetna Life, Hartford, Conn. |
| I. Read Stidger, M. D. | Prudential, Newark, N. J. |
| Arthur C. Stokes, M. D. | Guarantee Mutual Life, Omaha, Neb. |
| Archibald R. Stone, M. D. | Midland Mutual, Columbus, Ohio. |
| S. J. Streight, M. D. | Canada Life, Toronto, Ont. |
| Earl V. Sweet, M. D. | Mutual Benefit, Newark, N. J. |
| Bion C. Syverson, M. D. | Equitable Life Assurance, New York City. |
| Harold F. Taylor, M. D. | Aetna Life, Hartford, Conn. |
| William Thorndike, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| Walter E. Thornton, M. D. | Lincoln National, Ft. Wayne, Ind. |
| Ira G. Towson, M. D. | Fidelity Mutual Life, Philadelphia, Pa. |
| Wallace Troup, M. D. | Metropolitan Life, New York City. |

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| Frank L. Truitt, M. D. | Reserve Loan Life, Indianapolis, Ind. |
| Henry B. Turner, M. D. | Metropolitan Life, New York City. |
| John S. Turner, M. D. | Southland Life, Dallas, Texas. |
| Joseph P. Turner, M. D. | Jefferson Standard, Greensboro, N. C. |
| Henry G. Tuttle, M. D. | Metropolitan Life, New York City. |
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| Harry E. Ungerleider, M. D. | Equitable Life Assur., New York City. |
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| George A. VanWagenen, M. D. | Mutual Benefit, Newark, N. J. |
| Reynold C. Voss, M. D. | Pan-American, New Orleans, La. |
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| Albert A. Wagner, M. D. | Reliance Life, Pittsburgh, Pa. |
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| William R. Ward, M. D. | Mutual Benefit, Newark, N. J. |
| Faneuil S. Weisse, M. D. | Mutual Life, New York City. |
| Fred L. Wells, M. D. | Equitable Life Insurance, Des Moines, Iowa. |
| David E. W. Wenstrand, M. D. | Northwestern Life, Milwaukee, Wis. |
| Charles D. Wheeler, M. D. | State Mutual, Worcester, Mass. |
| Chester F. S. Whitney, M. D. | Home Life, New York City. |
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| Thomas H. Willard, M. D. | Metropolitan Life, New York City. |
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| Charles H. Willits, M. D. | Provident Mutual, Philadelphia, Pa. |
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| McLeod G. Wilson, M. D. | Travelers, Hartford, Conn. |
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| G. Elliott Woodford, M. D. | Home Life, New York City. |
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| Henry H. Schröder, M. D. | Verona, N. J. |

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| Berkshire Life, Pittsfield, Mass. | { Frank Harnden, M. D. B. W. Paddock, M. D. G. S. Reynolds, M. D. |
| California Western States Life, Sacramento, Calif. | H. W. Gibbons, M. D. |
| Canada Life Assurance Co. Toronto, Ont., Canada | { J. A. Roberts, M. D. H. C. Scadding, M. D. S. J. Streight, M. D. |
| Capitol Life, Denver, Colo. | F. W. Kenney, M. D. |
| Central Life Assurance Society, Des Moines, Iowa. | M. I. Olsen, M. D. |

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| Central Life of Illinois, Chicago, Ill. | |
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| Columbus Mutual Life, Columbus, Ohio. | W. B. Carpenter, M. D. |
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| Confederation Life Ass'n. Toronto, Ont. | { E. M. Henderson, M. D. F. W. Rolph, M. D. |
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| Connecticut Mutual Life, Hartford, Conn. | { C. D. Alton, M. D. C. B. Piper, M. D. H. B. Rollins, M. D. W. B. Smith, M. D. |
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| Continental Assurance Co., Chicago, Ill. | H. W. Dingman, M. D. |
| Continental Life, St. Louis, Mo. | C. R. Dudley, M. D. |
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| Dominion Life Assurance, Waterloo, Ont. | { A. J. McGanity, M. D. C. T. Necker, M. D. |

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| Great Southern Life, Houston, Texas. | J. E. Daniel, M. D. |
| Great West Life Assurance, Winnipeg, Man., Can. | W. L. Mann, M. D. |
| Guarantee Mutual, Omaha, Neb. | A. C. Stokes, M. D. |

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| Home Life, New York City. | { J. H. Humphries, M. D. C. F. S. Whitney, M. D. G. E. Woodford, M. D. |
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| Lincoln National Life, Fort Wayne, Ind. | { H. C. McAlister, M. D. W. E. Thornton, M. D. |
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| Manufacturers Life, Toronto, Ont. | { H. C. Cruikshank, M. D. R. C. Montgomery, M. D. |

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Baltimore, Md.

G. Carroll Lockard, M. D.

Massachusetts Mutual Life,
Springfield, Mass.

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Gordon Ross, M. D.
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Mutual Benefit Life,
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Waterloo, Ont.

{ J. M. Livingston, M. D.
R. L. Shields, M. D.

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THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA

1920-1933

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